THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—29TH YEAR. SYDNEY, SATURDAY, AUGUST 1, 1942. No. 5.

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WAR NEUROSES IN TOBRUK: A REPORT ON 207 PATIENTS FROM THE AUSTRALIAN IMPERIAL FORCE UNITS IN TOBRUK.

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and

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AT the beginning of May, 1941, a war neurosis clinic was established in an underground concrete shelter in Tobruk. Between May and August 207 men from the Tobruk fortress area were examined in this clinic. The records of these men have been followed through British and Australian hospitals and convalescent depots and through the medical records section of the Second Echelon, Australian Imperial Force. Some of the men still in hospital have been reexamined and those men still in the war neurosis clinic at Number 1 Australian Convalescent Depot have been interviewed. While the records are not complete, sufficient information has been obtained to form the basis of this report. In Tobruk detailed histories were recorded in the war neurosis ward. Although the shelter reverberated to shell fire and bombing, discussion with the patient could go on without the interruption of running to shelter. The capacity of the ward was 70 beds, and the patients could be retained in the war neurosis ward for a period of days or weeks. Prior to the opening of the war neurosis ward, both the hospital in Tobruk town and the section established on the beach had been repeatedly bombed. Patients were afraid to enter the hospital, as both patients and hospital personnel had been killed in various air raids. There was a tendency for men admitted to the wards suffering from medical or surgical conditions to develop a superadded neurosis in hospital. The opinion is expressed that a war neurosis treatment centre is necessary in the divisional area of operations. It should be stationed in a bomb-proof shelter and should have a capacity sufficient to hold patients for days or even a week or two. Its main function should be the classification of patients; this can be done only by medical officers experienced in this type of army medical work. No war neurosis patients should leave the divisional area except through the divisional neurosis clinic. One hundred and ten patients evacuated from Tobruk to the base have been traced. The records of these patients have been examined; they show that an appreciable delay arises between the patient's leaving the divisional area and his entering the war neurosis clinic which has been established at the Number 1 Australian Convalescent Depot. This delay in reaching the special treatment centre is to the disadvantage of the patient. Any treatment begun in the forward area is vitiated by delays in movement of patients. Treatment, to be of any use, must begin early and should be continuous, so far as this is possible. The movement of war neurosis patients, once they leave the forward area, has to be expedited through intermediate medical units so that the patient obtains early and continuous specialist treatment without delay. During a patient's stay in general hospital, new symptoms develop, old symptoms become fixed and repeated examinations by physicians and specialists establish in the mind of the patient an idea that he is suffering from obscure organic disease. Men with war neurosis which fails to respond to simple measures in the forward area must be treated in a special clinic, where the atmosphere should be more conducive to recovery. Occupational therapy, reeducation, explanation and other measures can be satisfactorily applied only in a place where the patient has absolute confidence in his medical officers, where the irksome restrictions of military life are reduced to a minimum and where the man can yet be trained once more to become an efficient soldier.

Here decisions can be reached as to the soldier's final classification as fit for front line service, for other duties, or for return to Australia.

Once a decision as to the further usefulness of the patient has been reached, there should be no delay in bringing him before a medical board. Once a man is "boarded", there should be no delay in his disposal.

The war neurosis clinic is firstly a unit for diagnosis and classification, and secondly a centre for the rehabilitation of those men who can be of further use in the army. Such a centre cannot be cluttered up with men "boarded" as unfit for further service; these should be held in camps well away from medical units.

The aim of treatment in the handling of war neurotics is threefold. The primary function of the medical services is the maintenance of the strength of the fighting force. No man should be evacuated from the divisional area who is capable of rendering further military service without adversely influencing his fellow soldiers. The second object is the rehabilitation of the subject as a soldier and as a man. If this rehabilitation can be carried out in the forward areas, so much the better. If the soldier has to return to the base for treatment in special clinics, this treatment must begin without delay. The third aim of treatment is to expedite the return of patients to civilian life when they can no longer serve as soldiers.

The majority of medical officers regard patients suffering from war neurosis as hopeless problems; the patient's condition is labelled "shell shock" or "bomb happy", and nothing is done to help him. Post-war pensions will be reduced if every officer takes an intelligent interest in the management of these patients.

The term "blackout", commonly used by war neurotics and medical officers to describe either anxiety attacks with dizziness or hysterically determined periods of stupor, is to be deprecated. The term bears a close resemblance to the "blackout" of high speed flying in aircraft and the tendency is for it to suggest physical disability in the face of physical stress. It is used complacently by hysterics as a rather distinguished badge of infirmity.

Of the 207 patients suffering from war neurosis treated in Tobruk, 79—that is, 38%—were returned to their units without leaving the fortress area. These men are still with their units at periods ranging from two to four months since their return to duty. An additional 48 men, after treatment at the base, have returned to duty with a classification of "A" (fit for front line service). From this it appears that 61% of patients with war neurosis admitted to hospital in Tobruk are now serving as fighting soldiers. An additional 48 men—that is, 23% of the total 207—are classified as fit for base duties ("B"). From the above facts it would appear that four out of every five men who break down under the stress of modern warfare are capable of further useful service.

Psychogenic Factors in Patients from Tobruk.

Many factors operate in the production of a neurosis in peace time, as in war. The pre-war personality of the soldier varies necessarily from one individual to another. The factors operating to provide a personality are constantly changing. Among the patients with a war neurosis seen in Tobruk, the psycho-biological make-up of every man was coloured for the time being by one dominating emotional reaction—fear. This fear was by most patients frankly appreciated and expressed.

In the majority of cases this reaction to fear was not permanent; it faded as soon as the massive stimulus producing fear was removed. In some cases there remained a morbid anticipation of bombing or shelling, which became a conditioned response. In others fear laid the basis for the development of an anxiety neurosis. As a patient moved through medical units away from the battle area further down lines of communication to a place of relative security, other considerations coloured the original fear reaction. Somatic anxiety equivalents appeared in some instances. In other cases, in which fear remained prominent, the soldier attempted to make his fear seem more reasonable by a simple dramatization and embellishment of his original experiences into something more impressive than real. Emotional over-reaction and over-statement of facts became pronounced. In some patients incapacitating symptoms such as exhaustion on the least exertion, insomnia, headache, anorexia and vomiting appeared or increased in degree. Physical and mental activity became limited. Pre-war maladjustments were now readily elicited when the patient's history was taken; application for return to Australia on compassionate grounds was made for the first time many weeks after the soldier reached base areas.

All the foregoing symptoms may have been simple additions to the original picture. They may have been produced unconsciously or they may have consciously been directed to the avoidance of a repetition of the original stress. On the contrary, these symptoms may have resulted from a simple uncovering of the more complete content of a neurotic state, originally masked by the dominating feature fear. The anxiety and terror inevitably associated with modern warfare are the exciting causes of psychoneurosis in almost every man who breaks down under stress. Among those patients who were seen many months after they left Tobruk, frankly expressed fear was less common and was often lost in a complex and confused picture of an established neurosis.

In Tobruk the personal and family history of every patient was recorded in detail. In the majority of cases, a prolonged stay in hospital resulted in the recording of several clinical histories before the patient finally reached the war neurosis clinic. Examination of successive records shows a variation of the original story; new symptoms arise, new words are added to the vocabulary. A single word spoken carelessly by a medical officer is seized upon, a question is interpreted as an opinion, and the history recorded at the war neurosis clinic is often materially different from that obtained in the forward areas.

It would appear that, before expressing an opinion as to the treatment and disposal of a neurosis patient, the medical officers at the war neurosis clinic should have the full medical record of that patient.

The medical officer in the forward area should, in so far as it is possible, obtain from the regimental medical officer and the patient's platoon or company commander all information as to the soldier's previous record in the unit, his behaviour under fire and the degree of psychic and physical trauma to which he has been subjected.

As in the neurosis seen in pre-war medical practice, the "previous personality" of the patient prior to the present stress was of considerable importance in the determination of the onset, the texture and the severity of the psychoneurosis. The personality of the patient was assessed after several interviews. The patient's version was in nearly all cases the only one obtainable, and as such was often misleading. Of the patients seen in Tobruk, the personality was regarded as of inferior type in 58% of those examined.

A history of previous "nervous breakdown" was obtained from 23% of the patients and of severe head injury sufficient to cause loss of conciousness prior to enlistment from 17%. In the majority of these cases the previous medical history had been concealed at the time of enlistment and did not appear on his "A.A. Form D1". A family history of neurotic traits was obtained from 50% of these 207 patients. From the above it would appear that more care in the questioning of recruits might result in a reduction of the proportion of men who break down under the stress of war.

The general health of the soldier, the degree of bombing to which he is subjected and the physical effects of "blast" all play some part in determining the onset of war neurosis. In Tobruk, food was adequate and of high quality; vitamins *B* and *C* were added to the diet. However, the monotony of an entirely tinned ration and the limited amount of fresh water available inevitably produced some effect. Exhaustion, other than that due to lack of sleep because of nightly bombing raids, was not a large factor in the psychogenesis of the majority of war neuroses seen in Tobruk.

Some men evacuated from the fortress area after battle injuries later developed a neurosis. However, the combination of physical disability and psychoneurosis appears

to be relatively uncommon, although no detailed figures for the Tobruk area have been obtained.

From the experience gained in Tobruk and the follow-up of patients suffering from war neurosis, certain opinions emerge. Accuracy and detail are essential in the recording of the history of the patient at all stages in his progress from his unit to the base. The whole medical record of a neurosis patient should be inspected before an opinion as to prognosis and treatment is expressed. While neurotic inheritance, previous breakdown, personality, exhaustion *et cetera*, play some part, the main existing factor in the production of war neurosis is fear.

An Analysis of 207 Patients seen in Tobruk.

Anxiety Neurosis.

The number of men suffering from anxiety and fear states exceeded those suffering from other forms of nervous disability. In all, 132 were regarded as suffering predominantly from anxiety states. The 70 men whose condition was labelled anxiety neurosis had the familiar somatic manifestations essentially similar to those seen in time of peace. In practically all these patients overt fear was present and the majority of them recognized that fear was the basis of their symptoms.

Patients presenting the picture of "effort syndrome" were not common. Their symptoms were uniform in character and differed from the other anxiety states. The impression formed was that of a physiological or pathological disorder which deviated from the orthodox conception of an anxiety state.

Under the title of fear states were placed those conditions in which bombing of no more than ordinary severity induced such a state of fear that either the soldier was incapable of continuing his duty or his influence was such as to be regarded as a menace by his officers. Sixty-two patients were labelled as suffering from fear states rather than neuroses because their fear was not unfounded. It was not dependent on past incidents so much as conditioned by rapidly recurring stimuli in the shape of continued bombing. There was ample evidence that their fears were based on reality, not on retrospective stimuli as in anxiety neurosis. There was an almost complete absence of the somatic manifestations of true anxiety neurosis.

At the first warning of an actual air raid, the patient with a fear state would run to cover. During the raid, these patients became pale; they had extreme, often audible, tachycardia, profuse sweating, pronounced tremor and, curiously enough, repeated yawning. A few patients showed motor restlessness, running from shelter to shelter. A desire to micturate was common; in some cases it was delayed until the raid had passed, in others involuntary micturition occurred.

These men, in a fear state, seemed to show an emotional disturbance which was no more than an exaggeration of the normal fear response. An undisciplined, animal-like fear occurred often in men with a poor personality or in patients suffering a concomitant physical illness such as dysentery.

Of the total 62 men suffering from fear states, 33 were sent back to their units in Tobruk, with a recommendation to the regimental medical officer that if they were unable to give satisfactory service somewhere in their unit, they should be returned to the base, but not through medical channels. All but five of these men are still serving as fighting soldiers. It would appear that 85% of men with simple fear states who are returned to their units after a week or two of rest in a war neurosis clinic within the divisional area, can be expected to give further service in the front line.

Of the 29 patients suffering from fear states who were evacuated to base hospitals, 19 had in addition some other physical or psychological defects, such as an old head injury, congenital mental defectiveness, *et cetera*.

Of all the men whose conditions were labelled fear states (62 in number), 39 are now classified as fit for front line service—that is, 63%.

Men with uncomplicated fear states were not evacuated to the base through medical channels. This was an attempt to prevent the easy escape from the fortress area of men who were merely frightened. As these men exhibited only extreme fear, it was considered desirable to label the field medical card (A.F. 3118) "fear state", in the hope that this diagnosis would not be bandied about post-war parlours, consulting rooms or assessment tribunals as readily as "neurosis", "blast" or "shell shock".

The men suffering from fear states were not regarded as medical casualties; although they were unable to stand up to the stress of front line service, evacuation to the base through medical channels would lay open an avenue of escape that might be increasingly used in a beleagured garrison, such as Tobruk. On the contrary, men suffering from anxiety neurosis in the majority of instances required specialist attention away from the fighting area. Forty-five were evacuated; 20 were returned to their units. Thirteen of the 20 who went to the base have since treatment been classified as "A" (fit for front line service). Three of those returned to their units in Tobruk have since broken down and have been reduced by medical boards to lower categories. In all, 35 out of a total of 70 men with anxiety neurosis have returned to front line duty. From the foregoing fact it appears that anxiety neurosis in soldiers has a relatively good prognosis if treated early: 73 men out of a total of 132 are classified "A" (55%) and 40 as "B" (30%).

Age is of importance in the prognosis of anxiety states; 90% of men with this condition who are under the age of thirty-five years should give further useful service in the army. Of the 132 men with anxiety states, 90 were under the age of thirty-five years; 55 of these are now serving with their original unit or are classified as "A" (61%), while 26 are classified as "B" (29%). On the contrary, of men aged over thirty-five years, 43% are classified as "A" and 33% as "B". Of the 13 men returned to Australia classified as "D" (permanently unfit), several were over the age of thirty-five years.

Every man under the age of thirty-five years who was returned to Australia with an anxiety state had defects of personality and of family history or had other psychiatric abnormalities; all but one of these men had suffered a "nervous breakdown" prior to enlistment.

Fear state, anxiety state and effort syndrome are subgroups of the larger class of anxiety neurosis, in all of which the prognosis is relatively good.

In both fear and anxiety states the soldier often overstated his case. By hysterical or conscious overlay, he dramatized the emotional situation during which he suffered the psychic trauma under which he broke down. This exaggeration was consciously or unconsciously designed to sway the medical officer's opinion, so that the patient could avoid further danger.

Conversion Hysteria.

Conversion hysteria was relatively rare in Tobruk. Of 207 men seen in the neurosis ward, 34 were regarded as suffering from simple conversion hysteria (16%), while anxiety states reached 64% of the total psychic disorders. The condition of a number of men with other psychiatric or physical abnormalities had an added hysterical colouring. Conversion hysteria occurred almost entirely in young men. Twenty-six of the 34 men with conversion hysteria are now with their units or classified as "A" (fit for service with field formation)—that is, 77%. One-half of these men returned to their units without leaving the fortress area. The majority of these patients responded quickly to crude suggestion.

As only one man with hysteria has returned to Australia, and 77% are classified as "A" and 20% are classified as "B", it would appear that the immediate prognosis of conversion hysteria is good.

The possibility of relapse in patients with conversion hysteria is great; as a common manifestation of hysteria in the army is a "fugue", these patients may be a menace in a unit if amnesia or other abnormality of behaviour occurred while the man was on duty. For this reason many such patients were evacuated to the base and classified "B", although at the time of examination by the medical board there was no residual disability.

In many cases it was impossible to determine where hysteria ended and where the conscious overlay and mimicry of the malingerer began.

Psychiatric Disturbances other than Anxiety States and Hysteria.

Five men showed hypochondriasis; these were all over the age of thirty-five years and their disorders were in no way different from the peace-time neurosis. There was the same inflexible insistence on the importance of symptoms and an unassailable preoccupation with visceral sensations. Other patients showed hypochondriacal additions to some other psychoneurosis; by the time these men reached the

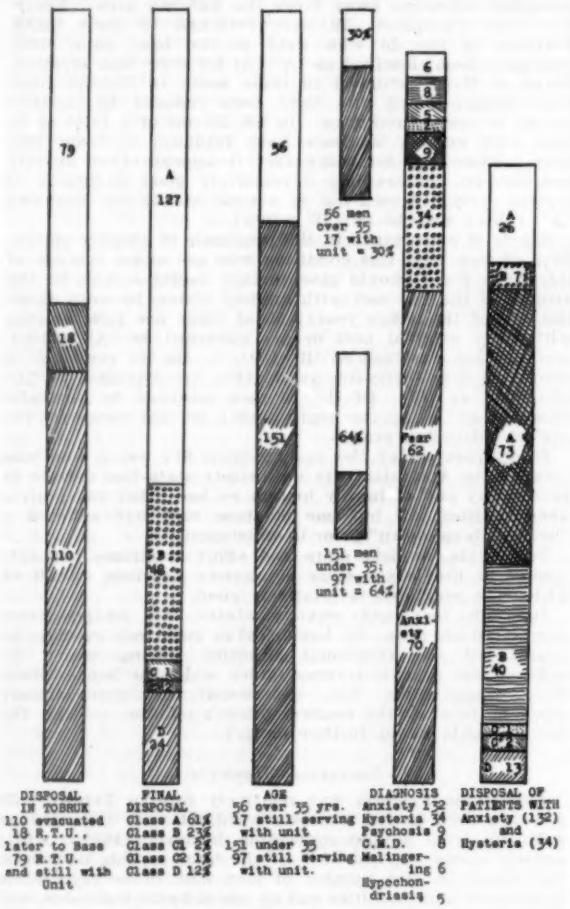

FIGURE I.

R.T.U.: "returned to unit"; C.M.D.: "congenital mental defect".

war neurosis clinic their primary defect was often forgotten and their visceral symptoms became predominant.

Psychopathy was uncommon; in these patients the personality defect was extreme, and they had been unable to settle down to a hard life or to army discipline. They became still more unsatisfactory soldiers in the face of fear, being incapable of any duty entailing the taking of responsibility and often reacting to superior authority by aggressive behaviour.

Congenital mental defectiveness was obvious in only ten instances; it varied in degree. None of these men should have been enlisted; all have returned to Australia.

Only six men were returned to their units with a diagnosis of malingering. This reflects great credit on the regimental medical officers in the fortress area, who culled the malingerers from the sick before they entered the hospital. In other cases (seven) there was probably a conscious motivation of symptoms, and doubt still exists as to the diagnosis in some. Conscious overlay upon other psychoneuroses increased the further the patient travelled away from the front line.

Remarkably few patients with frank psychosis were seen in Tobruk; only four cases of schizophrenia were seen among Australian Imperial Force patients, and there was one case of psychotic depression.

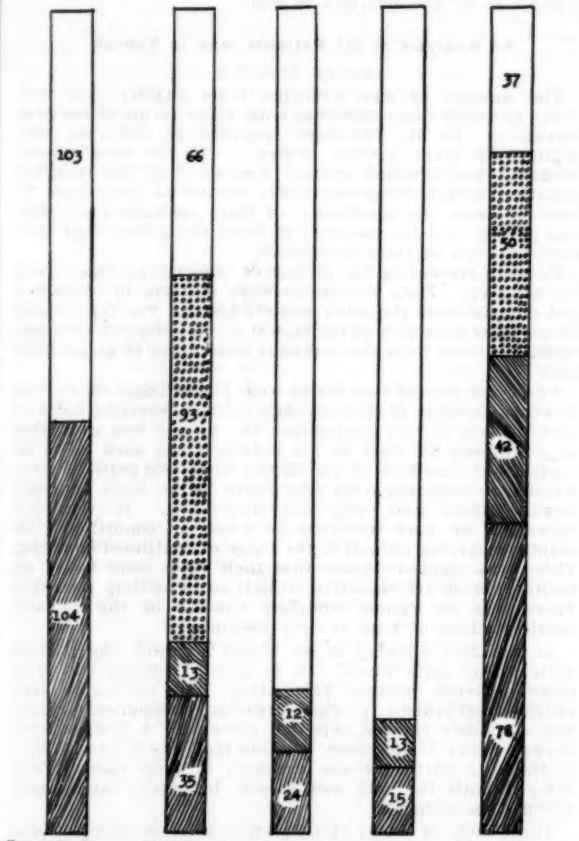

FIGURE II.

P.T.E.: "prior to enlistment".

Exhaustion States.

Considerable difficulty was encountered in estimating how great a part exhaustion played in the development of neurosis in Tobruk. Only two Australian Imperial Force patients were regarded as suffering from true exhaustion states. These men had been subjected to stress in excess of the average. They both showed improvement in a relatively short time.

Physical exhaustion of severe grade was uncommon in Tobruk. There was little movement of troops, and food supplies were always good. With the exception of the men manning the anti-aircraft defences and operating the harbour facilities, the emotional and physical exhaustion involved in bombing and shelling was more or less common to all in the fortress area. Lack of sleep due to nightly raids was the largest factor in the production of exhaustion.

Head Injuries.

A history of head injury prior to enlistment, of sufficient severity to render the patient unconscious, was elicited from 36 men (17% of the total 207 examined). By the large majority of these men this history had been concealed at the time of enlistment.

A soldier who had suffered a severe head injury did not stand up to the noise of shelling and bombing. These men developed headaches and other symptoms, and they were frequently admitted to hospital for investigations. In some cases there was a physical cause for the headaches; in others a psychoneurosis developed around the incident of the original injury. Many men unable to stand the strain of the front line used their previous injury as a means of escape from further service. A decision as to the nature of the headache is always a difficult one. No man should be accepted for service who has had a major head injury. Headache becomes more common as a symptom in base hospitals and at the war neurosis clinic. Vomiting was the most frequent symptom in Tobruk; headache is more common at Kfar Vitkin.

Treatment of Patients.

In the Unit.

The regimental medical officer of a unit should be able to deal with the majority of men who are in the early stages of anxiety neurosis or fear states. He can suggest that certain men be given duties away from extreme stress; he can talk to individual patients and in many instances he can prevent the development of anxiety states. The regimental medical officer can do a great deal to keep up the morale of a unit.

In Tobruk General Hospital.

When the psychiatric centre at Tobruk General Hospital was first opened, it was optimistically expected that the principle of treatment of early cases of war neurosis near the front line could be carried out in Tobruk. This was done so far as was possible, but as time went on it became more and more obvious that the psychiatric ward was, for the majority of patients a diagnostic centre.

The reasons for the failure of the ward to develop into a therapeutic centre were many. The ward was situated in a concrete shelter under an anti-aircraft gun and in the neighbourhood of a heavy anti-aircraft battery. The ward was therefore noisy, and there was difficulty in convincing a terrified patient that he was perfectly safe in the ward when the very sounds that conditioned the fear were, at times, more in evidence than in the front line.

The psychiatric ward was at first poorly lighted, by hurricane lamps for most of the period under review in this report. Recreational and occupational therapy was out of the question. There was bed room only, and the men slept in double-decker canvas barrack beds. Owing to the limited accommodation a prolonged stay in hospital was not possible; only men of good personality with hysterical states and fear states could be adequately treated in a few days or weeks. Finally, there was little opportunity to bring about gradual physical readjustment to conditions of warfare. Men on discharge from the hospital were required to return direct to their units or to a convalescent camp, which was often shelled or bombed.

Treatment, in so far as it could be carried out, consisted primarily in the production of adequate rest, if necessary, with the use of soporifics. Thereafter, there was a frank discussion of the nature of the patient's fear and of the distinction between fear and cowardice. If the personality of the patient was good enough, attempts were made to teach the patient to discipline his fear and to prove to him that he was capable of first-class work while still afraid.

It is regretted that more treatment was not carried out in the fortress area and that so many patients were evacuated to base hospitals. Successful psychotherapy should be possible nearer to the front line than at base hospitals or special hospitals.

In a War Neurosis Clinic at the Base.

A war neurosis clinic was established at Number 1 Australian Convalescent Depot by Major R. Whishaw. A detailed history can be recorded in such a clinic, pre-war defects can be discovered and a more accurate picture of the psychological make-up of the patient can be made. All previous army records of a patient's medical history are examined, and a decision is reached as to his future in the army or in a civilian sphere. Men "boarded" as unfit for further service are not retained in this clinic, but are returned to Australia, where they should receive vocational guidance as well as medical treatment.

Those men from whom further useful army service can be expected need reeducation and guidance as well as rest. The military atmosphere must be reduced to a minimum; but discipline must be retained, as these men have finally to return to army units. A war neurosis clinic calls for a psychiatrist, a psychologist, a vocational guidance officer, an occupational therapist and an army officer of experience. These qualities can rarely if ever be combined in one individual officer, so a war neurosis clinic needs a carefully selected team. The patients must be classified, advised, taught occupational therapy and given sufficient physical exercise to keep them fit and sufficient training to ensure that they are still soldiers. They must have sufficient time for recreation, and this recreation should be largely the result of the efforts of patients themselves. A concert by the patients is better than a cinema.

The vocational officer at the war neurosis treatment centre must be in close touch with a vocational or amenities officer in the base area, where men are distributed to units once they have completed their treatment at the war neurosis clinic. The vocational officer at the base should attempt to place men in jobs for which they are best fitted by training and temperament. Medical boards and psychiatric officers should indicate on the patient's papers the particular occupation for which they think a man is fitted. By this practice trends of usefulness, either developed or exposed during observation, would be followed up. The placing of "square pegs in round holes" so characteristic of the army should be obviated by the above measures.

Conclusion.

The prognosis of war neurosis depends largely on accurate diagnosis and early treatment. To an equal extent the results of treatment depend on the cooperation of the soldier's unit and the whole of the army organization. The results obtained in Tobruk and Kfar Vitkin justify the statement that the prognosis of war neurosis is good. Sixty *per centum* of the men have returned to units as fit for front line service, and only 12% have returned to Australia permanently unfit.

THE EFFECT OF IRRADIATION WITH ULTRA-VIOLET RAYS AND SHORT WAVES UPON METABOLISM AND INSENSIBLE PERSPIRATION.

By ARTHUR LIPPMANN, M.D.,
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WITH the introduction of heliotherapy and treatment with artificial light it became of interest to discover whether the light erythema altered the total metabolism and, if so, whether the change in metabolism was the chief factor in healing. All previous work has been done only by measurement of the heat production by determination of the oxygen absorption and carbon dioxide production. The earlier investigations dealing with the effect of sunshine (or with the sun cure at high altitudes) were not reliable, as too many complicating factors were involved—especially the effects of the infra-red rays of the sun, of the wind and of different atmospheric pressure. Interference due to heat also makes valueless experiments

with carbon arc lamps, which have a high output of infra-red rays. Kestner[9][10] and his co-workers were the first to experiment with mercury lamps, in which the heat rays are minimal, and they found a pronounced increase in oxygen consumption during irradiation; but the increase had vanished by the time the erythema appeared. On the other hand, Kestner[11] published observations on erythema due to solar radiation (in which the effects of heat rays could not be eliminated); in these no definite increase in oxygen consumption was reported. The results of Kestner in respect of the effects of ultra-violet irradiation could not be confirmed by Lippmann and Völker,[12] who occasionally found a slight rise in metabolism during the actual application of the rays, but not after the development of erythema—a conclusion which requires to be somewhat modified in view of the results of the present investigation. Lippmann and Völker assumed that the immediate effect was due not to irradiation, but to the cooling of the skin during the experiment. Loewy,[13] Campbell[14] and Mason[15] reported similar findings; but the most recent experiments by Lehmann[11] show that there is a 12% increase of oxygen consumption six hours after ultra-violet irradiation, by which time the erythema is pronounced, and that by the next day the basal metabolic rate has returned to normal. All these results are difficult to compare, because in none of the experiments was the temperature factor controlled and because the different authors measured the possible effect of irradiation at different intervals.

In recent years the work on metabolism has been improved, in so far as not only the heat production, but also a part of the heat loss—namely, the insensible perspiration—can be easily measured. By measurement of the loss of weight of a subject over a definite period the value for the insensible perspiration can be found with great accuracy by means of the very accurate Sauter balance. As Benedict[16][17][18] and later workers found, there is a definite relationship between the basal metabolic rate and the insensible perspiration, so that a good method is now available for controlling experiments on oxygen consumption, and at the same time an opportunity is afforded of further investigating the cause and effect of light erythema.

Insensible Perspiration.

The amount of insensible perspiration, as determined by measurement of the hourly loss of weight of the body, comprises the three different factors: loss of water from the lungs, loss of water from the skin, and the difference between the weight of carbon dioxide given off and oxygen absorbed. The last-named factor, amounting to only about three grammes per hour under basal conditions, can be kept constant and may therefore be neglected in the following experiments. The water loss from the respiratory passages can be kept constant to a high degree if the conditions of the surroundings are kept uniform. As the total amount of water lost from the lungs and from the skin is fixed under normal basal conditions, the most important variable is the water loss from the skin, which is the factor directly affected by irradiation. The total normal hourly amount of insensible perspiration is about 30 grammes and varies with the body surface and to a slight extent with the age of the subject. Two-thirds of the water is lost by the skin and about one-third by the lungs. In a previous publication[19] it was shown that the insensible perspiration of a given subject is a fixed quantity, provided that the subject is in basal condition and that the skin temperature is kept constant. If the "average" skin temperature (as explained in the article just referred to) is lowered by about 3° C., the insensible perspiration is decreased by about 15%. This "cooling effect" is pronounced and can always be reproduced. The temperature factor is therefore most important in all experiments, as only those experiments are comparable in which no noticeable cooling of the skin has occurred. Since it has been found that the measuring of the temperature of one skin area only (as, for example, the forehead) is without value, because different parts of the body surface react differently to cooling, at least eight different fixed points of the body have to be controlled and an average skin temperature has to be worked out from the temperature recorded and the size of the areas tested.

The oxygen consumption during the cooling experiments was also measured, and it was found that cooling of the skin to the extent of about 3° C. increased the oxygen consumption by about 5%—a fact which has usually been overlooked in experiments on erythemas.

Experiments with Ultra-Violet Irradiation.

Technique.

The subjects were mostly students, aged about twenty years, in good physical condition and accustomed to open-air sport. They had their last meal (protein-free) the previous evening, and came to the laboratory as a rule by motor car and rested for about an hour before the experiment. The experiment room was automatically maintained at a constant temperature of 23°–24° C. and the relative humidity was kept between 50% and 60%. The students were dressed in pyjamas and rested quietly on the Sauter balance, covered with a sheet. All sheets used had previously been kept in the room to eliminate all possibility of variation in humidity. As soon as the weighing controls indicated that the conditions had become stable, the experiment was continued for about an hour. The times required for the loss of each 5 grammes of body weight were recorded and from these figures the amount of the hourly insensible perspiration was calculated. The basal metabolic rate was determined at the end of this period with the Krogh or the Benedict apparatus, and the skin temperatures were controlled. After this, the clothing was removed and ultra-violet irradiation by means of a mercury vapour lamp was applied, first to the front and then to the back of the body, each for a period of four minutes and at a range of one yard. This was sufficient to produce a pronounced erythema. The subject, who remained passive, was then dressed and covered and the weighing experiment was continued for at least two hours, after which the basal metabolic rate was determined and the skin temperature recorded. The following morning the experiment was repeated under the same conditions. Only experiments in which the average skin temperature changed less than 1° C. during the weighing experiment have been recorded.

Ultra-Violet Erythema.

The ultra-violet erythema was first noticed about ninety minutes after exposure, and was nearly always distinct after a further twenty minutes; it became very florid in the further course of the day and was still pronounced the next morning. It was observed that the temperature of the exposed skin had already begun to rise within fifteen minutes after the irradiation and that it was about 0·7° C. higher thirty to seventy minutes after the exposure, at a time when not the slightest sign of a skin reaction could be seen. As a rule there was no further rise of skin temperature with the appearance of the erythema; only in a few cases was the appearance of the erythema accompanied by a further increase in temperature to 1·2° or 1·5° C. above that of a non-irradiated area of skin. The difference in temperature between irradiated and non-irradiated skin was maintained until the following day or even for a longer period—in some cases for at least three days. A similar difference in the skin temperature has previously been observed by Schwanhausser and Bayer.[20] The relative difference between the temperature of irradiated and non-irradiated skin persists even after cooling, when the level of the temperature of the skin as a whole is lowered to the extent of about 3° C. and the difference may even be accentuated by such cooling.

Oxygen Consumption.

In nearly all cases a small increase in oxygen consumption was observed two hours after the subject's exposure to ultra-violet rays—that is to say, at a time when a pronounced erythema had developed all over the body and was in progress. This increase was still present after a period of twenty-four hours, but occasionally by this time the values had begun to fall again towards the normal level.

Insensible Perspiration.

More interesting are the facts concerning the insensible perspiration. In the first twenty minutes after the exposure to the rays there was usually a small increase, perhaps as a result of the slight amount of movement during dressing after the irradiation experiment. Beginning thirty minutes after irradiation and approximately simultaneously with the above-mentioned rise of the skin temperature, there occurred a pronounced decrease in the insensible perspiration, and after the end of two hours a nearly constant average decrease of about 10% was noted. In most cases the insensible perspiration had not returned to normal after twenty-four hours; as a rule it was still slightly below normal at this period, while in some cases it was higher. It may be pointed out that this reaction cannot be explained as the result of cooling, for the body temperature as recorded in the mouth remained at a constant level, while the average skin temperature was actually somewhat higher (about 0·7° C.) than at the beginning of the experiment—an effect which is easily explained by the presence of erythema. Should it be objected that the changes in the basal metabolic rate and the insensible perspiration might be due to the prolonged rest of the subjects under basal conditions, it might be pointed out that control experiments without irradiation never revealed these changes; on the contrary, in the case of a subject who rested for longer periods, the oxygen consumption fell and the insensible perspiration remained constant provided that no cooling occurred.

Of the total water loss recorded as insensible perspiration, about 15% is due to the insensible secretion of sweat by the sweat glands of the axillæ, palms and soles. As these parts received no irradiation at all, the whole effect can be attributed to changes in the skin alone.

Discussion.

Since dilatation of the skin vessels brought about by heating with hot-water bottles or by the administration of drugs (yohimbine, amyl nitrite) is accompanied by an increase in insensible perspiration, it might have been supposed that a similar increase would occur as a result of an erythema induced by irradiation. As the above experiments show, this was found not to be the case. There must therefore be special factors acting in ultra-violet erythema which give rise to reactions different from those resulting from vaso-dilatation brought about by other means. From Haxthausen's[11] and from Ipsen's[13] work we know that the colour of the skin ("red" or "pale") is dependent almost entirely upon the state of the skin capillaries, which in a red skin are engorged with blood. But the skin temperature does not depend upon the amount of blood in the skin capillaries. The skin temperature does, however, depend on the condition of the arterioles in the deeper layers of the skin, which regulate the amount of blood passing through the skin by means of contraction or dilatation. In erythema resulting from irradiation the capillaries are certainly extremely engorged with blood (as shown by the red colour); but it may be assumed that there is also a dilatation of the arterioles, for, as was stated earlier, the skin temperature is raised. Indeed, this dilatation of the arterioles and increase of blood flow would seem to occur even before the appearance of erythema, as is suggested by the fact that the rise of temperature precedes the erythema.

A statement by Malten[12] can be explained in a similar way. Instead of measuring the skin temperature, he measured by means of a simple device the heat loss from irradiated and non-irradiated areas of skin. He found that the heat loss from erythematous skin was 25% higher than that from non-irradiated normal skin, and that while the heat loss from the irradiated skin remained unchanged after cooling, that from the non-irradiated skin diminished. This would seem to indicate that the arterioles in the irradiated skin are dilated and that they have lost their ability to contract in response to cooling. On the other hand, my findings show that while after cooling has occurred the temperature of the erythematous areas remains higher than that of the surrounding skin, it does fall somewhat; this indicates that the arterioles still retain some capacity to contract.

Cooling Experiments.

In order to discover to what extent the reactivity of the vessels in the erythematous skin is changed as a result of cooling, the following experiments were performed. In former experiments, published elsewhere,[10] it was shown that by cooling of the body of a healthy reactive person to the extent that the average skin temperature decreases (about 3° or 3·5° C.) the oxygen consumption was increased about 5% and the insensible perspiration was decreased about 10% to 20%.

These experiments were repeated on persons subjected to irradiation. The technique of the experiment was the same as that described above. After the data for the second day given in Table I had been obtained (that is, the figures for the subject kept in a comfortable surrounding temperature and with normal skin temperature), the experiment was continued after removal of part of the clothing and coverings for about an hour. The result was a decrease of the average skin temperature by about 3° C. After about an hour the insensible perspiration and basal metabolic rate were measured once again. The results are to be seen in Table II. As a rule it was found that the normal reaction to cooling persisted—that is to say, the oxygen consumption was a little increased, while the insensible perspiration decreased about 10% to 22%. If anything, the values obtained are rather higher than those formerly obtained from "non-irradiated" subjects; that is to say that a person having an ultra-violet erythema reacts to cooling to at least the same degree as an untreated subject. We know that the normal individual reacts to moderate cooling by a reflex action upon the thermogenic centres through the nerves conveying thermal sensibility. These centres respond by causing increased

TABLE I.

Oxygen Consumption and Insensible Perspiration after Irradiation with Ultra-Violet Rays.

Subject.	Age. (Years.)	Body Surface. (Square Metre.)	Average Skin Temperature. (Degrees Centigrade.)		Calories per 24 Hours.					Insensible Perspiration. Total Grammes per Hour.				
			Before Experiment.	2 Hours after Irradiation.	Before Irradiation.	2 Hours after Irradiation.	Percentage Change.	24 Hours after Irradiation.	Percentage Change.	Before Irradiation.	2 Hours after Irradiation.	Percentage Change.	24 Hours after Irradiation.	Percentage Change.
Be. ..	19	1·89	32·4	32·9	1,728	1,872	+8	1,775	+3	41·8	36·6	−12	40·3	−3
We. ..	26	1·84	33·7	34·1	1,525	1,557	+2	1,545	+1·5	34·3	30·3	−10	30·5	−10
L. ..	35	1·64	32·8	33·1	1,218	—	—	1,365	+11	27·0	23·4	−13	23·8	−13
G. ..	23	1·78	33·0	32·7	1,581	1,536	−3	1,620	+2	36·0	33·0	−10	38·2	+6
S. ..	46	1·91	32·4	32·4	1,420	1,444	+1·5	—	—	34·5	32·1	−8	—	—
Ru. ..	18	2·05	31·0	31·7	1,835	1,930	+5	1,923	+5	39·1	36·4	−7	42·3	+8
Bo. ..	23	1·79	32·8	31·8	1,640	—	—	1,732	+5	38·4	36·1	−5	37·5	−3
Ba. ..	22	1·78	32·4	33·2	1,616	1,828	+13	1,645	+2	31·9	30·0	−6	30·8	−3
Wu. ..	21	1·78	33·0	33·8	1,578	1,700	+8	1,553	0	37·8	35·5	−5	38·7	+2
Ro. ..	22	1·82	32·0	32·9	1,278	1,374	+8	1,397	+10	29·0	27·0	−6	29·4	0
A. ..	19	1·78	32·2	33·1	1,725	1,816	+5	1,721	0	31·1	29·5	−4	30·0	−3
A. ..	25	1·90	32·8	33·3	1,816	1,898	+4	1,814	0	38·1	38·4	0	38·0	0
E. ..	17	1·90	32·4	33·4	1,731	1,841	+7	2,003	+16	33·0	33·0	0	36·8	+10
H. ..	22	1·98	33·7	33·9	1,879	1,987	+6	2,100[1]	+11	39·0	36·8	−5	40·8[1]	+4

[1] Seventy-two hours after irradiation, instead of twenty-four.

TABLE II.[1]
Cooling Experiment.

Subject.	Age. (Years.)	Average Skin Temperature in Degrees Centigrade.			Mouth Temperature in Degrees Centigrade.			Calories per Twenty-four Hours.			Insensible Perspiration in Grammes per Hour.		
		Before Cooling.	After Cooling.	Decrease.	Before Cooling.	After Cooling.	Difference.	Before Cooling.	After Cooling.	Percentage Difference.	Before Cooling.	After Cooling.	Percentage Difference.
G. ..	23	33·2	30·3	−2·9	36·6	36·5	−0·1	1,620	1,778	+10	38·2	32·0	−17
Wh. ..	21	33·9	30·1	−3·8	36·1	36·1	0	1,553	1,740	+12	38·7	33·0	−14
R. ..	22	32·5	30·6	−1·9	36·0	35·6	−0·4	1,397	1,512	+9	29·4	24·9	−15
L. ..	35	33·0	30·6	−2·4	36·4	36·3	−0·1	1,365	1,349	−1	23·8	17·7	−22
We. ..	26	33·3	29·2	−4·1	36·4	36·2	−0·2	1,545	1,523	−1	30·5	27·3	−10

[1] The experiments were started twenty-four hours after intensive ultra-violet irradiation, at a time when pronounced erythema was present. The figures are those obtained before and after one hour's cooling.

heat production, as evidenced by increase of oxygen consumption, and at the same time diminished heat loss by vasoconstriction of the skin vessels; this results in a decrease of radiation and at the same time, as has been shown, a diminution in the insensible perspiration. The same reaction occurs in subjects with ultra-violet erythema. This shows that there is no interference with the reflex mechanism of heat regulation. Since the reflex mechanism is intact, any departure from the normal in respect of insensible perspiration can be due only to the local effect of the irradiation on the skin.

Discussion of Local Effects of Ultra-Violet Irradiation.

From the work of Krogh, Lewis, Ellinger[(6)] and others we know that histidine is always present in the skin cells, and this under the influence of the ultra-violet rays is transformed into histamine with loss of carbon dioxide. This very reactive compound diffuses quickly into the deeper layers and acts on the arterioles, causing dilatation. At the same time the extensive destruction of epidermal cells by ultra-violet irradiation gives rise to decomposition products, which diffuse much more slowly and act upon the capillaries, producing erythema. It is therefore possible that the substance causing arteriolar dilatation may not necessarily be identical with that responsible for the dilatation of capillaries, although histamine can alone produce both of these effects. This possibility is also suggested by the observation that the increase in skin temperature, which is due to arteriolar dilatation, may precede by as much as ninety minutes the appearance of erythema. We also know that erythema is the visible sign of the onset of inflammation of the skin, and this is accompanied by capillary engorgement and œdema. It is therefore to be anticipated that under these conditions the passage of water through the skin may be interfered with as a result of œdema, owing to pressure effects, and this would account for the observed decrease in insensible perspiration during the acute stage of erythema. This hypothesis receives support from the observation of Moog,[(20)] that subcutaneous injection of histamine causes similar changes—namely, erythema accompanied by a rise in skin temperature and at the same time a fall in the insensible perspiration in the affected area.

Experiments with Short Waves.

The possibility exists that the electric energy absorbed during ultra-violet irradiation can directly influence the heat regulation in some way other than by changes in the skin—a possibility which seems to be rather remote, as the power of penetration of ultra-violet rays is restricted. It was therefore considered desirable that the effect of some form of penetrating rays should be studied.

It is known that short waves have very little effect upon the skin (except heating effect), but that they are capable of penetrating the entire thickness of the body. It is conceivable, therefore, that they may influence the deeper nerves or may act by heating the blood and by both of these means indirectly influence the heat-regulating centres.

Previous experimenters (Schliephake[(30)]) in this field have worked with short-wave irradiation in very high doses, sufficient to produce a pronounced heating of the body as a whole (electropyrexia), resulting in sweating and considerable increase in oxygen consumption. The interesting observation has been made that after irradiation of one-half of an animal's body, with resulting heating of the part, the temperature of this half subsequently falls to a level below that of the non-irradiated side; this indicates some form of local over-compensation. Pflomm[(28)(29)] showed that the permeability of the capillaries after the application of short waves is much decreased—a result which also calls for experiments on insensible perspiration.

With the exception of experiments on rats, I could find no reference to any previous work in this field. Rats showed a pronounced discharge of water vapour during the irradiation, but most of the water was certainly discharged by sweating.

Technique.

As it was desired to demonstrate how the insensible perspiration is affected by the application of short waves, it was necessary carefully to avoid all sweating. The dose of short waves that could be employed was therefore restricted. As the possibility existed that the heating effect of short waves might not be the only important factor producing therapeutic effects, but that there might be still other direct and specific effects of the short waves on the body, it was thought that the application of smaller doses might be sufficient to produce definite results. It was considered desirable to avoid all direct action on the thermogenic centres; therefore only the trunk was irradiated, one electrode (36 square centimetres) being placed on the back, the other on the abdomen. Undoubtedly there is a certain local heating of the affected skin, which becomes red for about ten minutes; but the area directly exposed is small compared with the whole body surface, and no case in which local or general sweating occurred was recorded. A sparking machine was used, the wavelength employed being usually between nine and twelve metres. Owing to the fact that the energy loss during transmission from the apparatus to the body was an unknown factor, it is not possible to state exactly the amount of energy absorbed by the body. Preliminary experiments were, however, performed to find the maximum dose which could be applied to the subject without the induction of sweating, and this dose was employed in the subsequent experiment.

Twenty-three experiments were carried out on twelve students. The technique was the same as in the ultra-violet irradiation experiments—that is to say, to persons whose insensible perspiration, oxygen consumption and average skin temperature had been measured during a control period, short waves were administered for twenty minutes, and immediately after this the oxygen consumption was measured and the insensible perspiration was recorded for a period of seventy to ninety minutes; then the basal metabolic rate and the temperature were again measured.

TABLE III.
Experiment with Short Waves.

Subject.	Age. (Years.)	Surface. (Square Metres.)	Time of Experiment. (Minutes after Irradiation.)	Average Skin Temperature. (Degrees Centigrade.)			Mouth Temperature. (Degrees Centigrade.)			Calories per Twenty-four Hours.					Insensible Perspiration in Grammes per Hour.				
				Before Irradiation.	At End of Experiment.	Difference.	Before Irradiation.	At End of Experiment.	Difference.	Before Irradiation.	Immediately after Irradiation.	Percentage Difference.	At End of Experiment.	Percentage Difference.	Before Irradiation.	Immediately after Irradiation.	Percentage Difference.	At End of Experiment.	Percentage Difference.
St.	21	2·00	70	32·6	32·5	−0·1	36·6	36·6	0	1,695	1,737	+2	1,779	+5	40·4	39·0	−2	34·1	−15
F.	21	1·64	90	32·8	33·0	+0·2	36·7	36·7	0		1,563		1,598		30·9	27·6	−10	25·9	−16
M.	21	1·80	80	32·6	32·8	+0·2	36·7	36·5	−0·2	1,751	1,751	0	1,668	−5	37·5	32·8	−13	30·0	−20
E.	22	1·98	80	32·5	31·9	−0·6	36·9	36·8	−0·1	2,001	2,022	+1	1,897	−5	44·3	38·8	−12	40·5	−10
W.	21	1·78	90	33·0	32·3	−0·7	36·4	36·6	+0·2	1,440	1,406	−2	1,580	+10	27·0	27·4	0	24·0	−9
Re.	21	1·97	60	34·2	34·1	−0·1	36·9	36·9	0	1,894	1,967	+4	2,030	+7	36·0	39·5	+10	33·0	−8
Ro.	21	1·92	75	33·4	33·6	+0·2	36·8	36·4	−0·4	1,779	1,911	+7	1,848	+4	40·8	41·9	+2	43·0	+5
A.[4]	20	1·78	90	32·9	32·9	0	36·6	36·7	+0·1	1,772	1,827	+3	1,841	+4	26·0			27·0	+4
O.	21[1]	1·87	45	33·7	33·3	−0·4	36·6	36·4	−0·2	1,737	1,945	+12	1,737	0	36·8	31·0	−16	37·0	0
	[2]		90	33·0	33·0	0	36·4	36·5	+0·1		1,668	+3·5	1,737	0	38·8	37·6	−2	34·0	−12
	[3]		60	33·5	33·8	+0·3	36·6	36·6	0	1,813	1,765	+3	1,692	−7	37·5	46·0	+23	50·0	+25

[1] Weak dose for fifteen minutes only. [2] Normal dose [3] High dose; sweating. [4] Diabetes. I.P. figure low.

Results.

As can be seen from Table III, the body temperature could be kept nearly constant; the changes in body temperature were so insignificant that no changes in insensible perspiration could be ascribed to this factor. As a rule there was an increase in oxygen consumption, which, although rather small, was significant, because it was nearly constant in all cases. At the same time, in a proportion of cases the insensible perspiration began to decrease immediately after the irradiation, while in practically all cases it was decreased (to an extent of about 10%) at the end of one hour.

These findings are similar to those described earlier as resulting from the ultra-violet irradiation. This is stated only as a fact of observation; it is not assumed that the mechanism of response is necessarily the same to both kinds of irradiation. Whereas substantial evidence has been advanced to show that changes in insensible perspiration resulting from the action of ultra-violet irradiation can be ascribed to local changes in the skin, no such certainty exists regarding the response to short-wave irradiation. The latter may conceivably act upon the deeper structures.

Summary.

1. After irradiation with ultra-violet rays, the irradiated skin reacts almost immediately by a rise of temperature, which is noticeable one hour before an erythema is visible.

2. Irradiation with ultra-violet rays results in a decrease in insensible perspiration, which precedes the onset of erythema and becomes pronounced with the development of erythema. Two hours after irradiation—by which time the erythema has become pronounced—the oxygen consumption is found to be a little increased and has not returned to normal on the following day, while the erythema persists.

3. Cooling experiments show that the reflex mechanism (through the nerves conveying thermal sensibility and the heat-regulating centres) of constriction of the skin arterioles is unaffected by ultra-violet irradiation. It is concluded that the changes in insensible perspiration are due to local changes in the skin.

4. Short waves applied in doses insufficient to bring about pyrexia or sweating in most cases cause a rise in oxygen consumption and a diminution in insensible perspiration, the changes becoming definite after eighty minutes. These reactions occur in the absence of local changes in the skin.

Acknowledgement.

I wish to thank Professor Lambie for his help and advice and for granting me all necessary facilities in the department of medicine. I also desire to thank the Senate of the University of Sydney for a grant which enabled me to undertake this investigation.

References.

(1) F. W. Bayer and E. V. Philipsborn: *"Der kutane lokale Abkühlungsversuch nach Ipsen als Funktionsprüfung der Arteriolen", Strahlentherapie,* Volume LXIII, 1938, page 129.

(2) F. G. Benedict and C. G. Benedict: "Insensible Perspiration: Its Relation to Human Physiology and Pathology", *Archives of Internal Medicine,* Volume XXXVIII, 1926, page 1.

(3) F. G. Benedict and C. G. Benedict: *"Perspiratio insensibilis, ihr Wesen und ihre Ursachen", Biochemische Zeitschrift,* Volume CLXXXVI, 1927, page 278.

(4) F. G. Benedict and H. S. H. Wardlaw: "Some Factors Determining the Insensible Perspiration of Man", *Archives of Internal Medicine,* Volume XLIX, 1932, page 1019.

(5) J. A. Campbell: "Ultraviolet Radiation and Metabolism, with a New Method for Estimating Metabolism", *Proceedings of the Royal Society of Medicine,* Series B, Volume XCIX, 1926, page 451.

(6) F. Ellinger: *"Ätiologie des Lichterythems", Strahlentherapie,* Volume XL, 1931, page 760.

(7) H. Haxthausen: *"Einige Untersuchungen über die Kreislaufveränderungen in der Haut nach Bestrahlung mit verschiedenen Lichtquellen", Strahlentherapie,* Volume XXX, 1928, page 662.

(8) O. Kestner, F. Peemöller, R. Plaut: *"Die Einwirkung der Strahlung auf den Menschen", Klinische Wochenschrift,* Volume II, 1923, page 2018.

(9) O. Kestner, F. Danmeyer, F. Peemöller, R. Liebeschuetz-Plaut: *"Die Heilwirkung des Höhenklimas", Klinische Wochenschrift,* Volume IV, 1925, page 910.

(10) O. Kestner, C. E. Johnson, W. Laubmann: *"Stoffwechsel und Sonnenstrahlung", Strahlentherapie,* Volume XLI, 1931, page 174.

(11) G. Lehmann: *"Einfluss der Ultravioletbestrahlung auf den Arbeitsstoffwechsel und die Arbeitsfähigkeit des Menschen", Strahlentherapie,* Volume XLVIII, 1933, page 364.

(12) A. Lippmann: "On the Insensible Perspiration and its Clinical Significance", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1942, page 569.

(13) A. Lippmann and H. Völker: *"Beiträge zur Frage der Stoffwechselbeeinflussung durch Ultravioletbestrahlung", Klinische Wochenschrift,* Volume V, 1928, page 213.

(14) A. Loewy: *"Über die physiologische Wirkung der Ultravioletbestrahlung", Strahlentherapie,* Volume XXIX, 1928, page 333.

(15) H. Malten: *"Wärmeregulation und Lichtwirkung", Münchener medizinische Wochenschrift,* Volume LXXIX, 1932, page 1472.

(16) E. H. Mason and H. H. Mason: "Effect of Ultraviolet Light on Oxygen Consumption and on Total Metabolism", *Archives of Internal Medicine,* Volume XXXIX, 1927, page 317.

(17) O. Moog: *"Die Bedeutung der Haut für die unmerkliche Hautwasserabgabe", Verhandlungen der deutschen Gesellschaft für innere Medizin,* Volume XXXVI, 1924, page 146.

(18) E. Pflomm: *"Kurzwellenbestrahlung des Rattensarkoms", Münchener medizinische Wochenschrift,* Volume LXXVII, 1930, page 1854.

(19) E. Pflomm: *"Experimentelle und klinische Untersuchungen über die Wirkung ultrakurzer elektrischer Wellen auf die Entzündung". Archiv für klinische Chirurgie,* Volume CLXVI, 1931, page 251.

(20) E. Schliephake: *"Kurzwellentherapie. III. Auflage",* 1936.

(21) K. Schwanhausser: *"Verhalten der Vasomotoren bei normaler und erythematöser Haut". Schweizerische medizinische Wochenschrift,* Volume LXIV, 1934, page 707.

Reports of Cases.

TWO CASES OF WEIL'S DISEASE IN WIDELY SEPARATED SUBURBS OF SYDNEY.

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Case I.

I FIRST examined H.C., aged forty-three years, on March 4, when he complained of severe pain in both thighs and calves and in the back of his neck, and also of severe headache and a slightly sore throat. He said that he had felt quite well when he ceased work that day, about four hours earlier, but whilst he was returning home he was suddenly stricken with these pains in his limbs and back. On clinical examination he had a slightly reddened throat and some generalized muscular tenderness. His temperature was 101·6° F. and his apex beat was in the fifth intercostal space, four and a half inches from the mid-line; a harsh mitral systolic murmur was present.

He had suffered from bronchitis for some years past, and in 1921 had been radiologically examined, as he then complained of dyspepsia. No ulcer was detected. He had had no other illnesses, but had sustained an injury to his right eye during the last war (1914-1918) and lost the sight of it.

Next day (March 5) his wife telephoned to me to say that he seemed much better. On March 6 he appeared very ill. His temperature was 103·6° F., his pulse rate was 120 per minute and the rhythm was slightly irregular. He had a few bronchitic signs in his chest and his tongue was dry and heavily furred. Labial herpes was present.

On March 7 his skin was slightly jaundiced and his conjunctivæ were injected and also slightly jaundiced. His tongue was still furred and dry, and he vomited dark brown fluid at frequent intervals. The muscular tenderness seemed less pronounced. His urine contained bile and his pulse rate still remained at 120 per minute; the blood pressure was 140 millimetres of mercury, systolic, and 90, diastolic; his temperature was 101° F.

On March 8 he still looked very ill. His pulse rate was 120 per minute, his temperature was 99·4° F. and the jaundice was more pronounced.

On March 9 the jaundice was deeper and the urine contained bile and one-twelfth of its volume was albumin. A morbilliform rash appeared, first as a few discrete patches on the abdomen, then over the remainder of his trunk, most pronounced on the back; it was very irritable. The lower border of the liver was tender and palpable one inch below the costal margin.

On March 10 a blood count by Dr. Eva Shipton gave the following information: the erythrocytes numbered 4,200,000 per cubic millimetre and the hæmoglobin value was 13·6 grammes per 100 cubic centimetres; the leucocytes numbered 16,000 per cubic millimetre, 82% being neutrophile cells, 16% lymphocytes and 2% monocytes. There was no shift to the left, but toxic changes were present.

On March 13 blood was taken for culture and guinea-pig inoculation, but no leptospiræ were seen (tenth day of disease).

On March 14 the temperature was 99·6° F. and the jaundice was slightly less; the patient appeared to be much better.

On March 20 his general condition was much improved. His temperature was normal, his pulse rate was 90 per minute and the jaundice was less. Urine was taken for culture, but although the specimen was alkalinized immediately after having been voided, no leptospiræ were seen. Jaundice was still present in slight degree during the fifth week of the patient's illness.

The patient's condition gradually improved. He was allowed up during the seventh week of his illness, and he was well enough to go to the University of Sydney for further urine culture and agglutination tests on April 24.

The urine was again examined on April 29 after intensive administration of alkali during three days, again with negative findings.

Agglutination tests, however, gave the following results:

Leptospira pomona (+++) 1:10, (++) 1:30, (+) 1:100, (trace) 1:300.

Leptospira australis A (+++) 1:10.

Leptospira australis B (+++) 1:10, (+++) 1:30, (++) 1:100, (++) 1:300, (+) 1:1,000.

Leptospira Mackney (+++) 1:10.

Leptospira icterohæmorrhagiæ (+++) 1:10, (+++) 1:30, (+++) 1:100, (+++) 1:300, (+++) 1:1,000, (++) 1:3,000, (+) 1:10,000.

Case II.

I am indebted to Dr. E. P. Armstrong for the following particulars of his patient (B.H.W.), who was working on the same closed drain as H.C. from February 23 to March 2. B.H.W. was first examined on March 7 and gave a history of vomiting two days previously, followed by general malaise and severe headache. When he was first examined he complained of severe headache, sore throat and generalized aches and pains. His temperature was 104° F. and he was sweating profusely; his tongue was furred and his pharynx was inflamed.

On March 8 he still complained of severe headache, and his temperature was 103° F. Jaundice appeared on March 11 and persisted for three weeks. He returned to work on June 8.

Examination of his blood and urine during the last week in April revealed no leptospiræ, but agglutination tests gave the following information:

Leptospira icterohæmorrhagiæ (+++) 1:3,000, (+) 1:10,000.

Leptospira australis A (+++) 1:10, (trace) 1:30.

Leptospira australis B (+++) 1:300, (++) 1:1,000, (++) 1:3,000, (+) 1:10,000.

Comment.

Both men had been working in the same drain one week prior to the onset of their illness. They reported that they noticed at the time several dead rats lying about. They were working in a closed drain, which had become obstructed during the latter end of the recent dry spell in Sydney.

The illness had the following notable features: (i) the sudden onset, accompanied by severe headache, muscular pains and pharyngitis; (ii) the severe prostration; (iii) the appearance of jaundice four and six days respectively after the onset of the illness; (iv) the labial herpes (not hæmorrhagic), morbilliform rash and hepatic enlargement (in one of the cases); (v) the absence of splenomegaly; (vi) the absence of a relapse, though convalescence was long in each case.

It was disappointing that no leptospiræ were obtained from the blood or urine in either case, though several examinations were made. However, the agglutination results leave no doubt as to the diagnosis.

Acknowledgement.

I wish to thank Dr. W. C. Sawers, of the School of Public Health and Tropical Medicine, University of Sydney, for the interest taken and the help given in establishing the diagnosis.

Reviews.

A SYNOPSIS OF HYGIENE.

IN spite of the war and its attendant difficulties of preparation and publication, a seventh edition of that very useful handbook Jameson and Parkinson's "Synopsis of Hygiene" has made its appearance this year.[1]

Unlike its predecessor, which was published in 1939, it embodies no great change in subject matter or make up, but the sections on infectious diseases, food, and water supplies have been brought up to date, and a considerable number of references to recent literature have been included. The sole concession to the existence of the war is a very brief section, two pages, on emergency measures. The chapter on personal hygiene is particularly good and contains a wealth of data which are as valuable to the general practitioner as to the health officer.

As is usual with overseas publications on public health and hygiene, there is much that is not applicable to Australian conditions—all the legal information for instance—but when due allowance has been made for this the book remains a most useful work of reference well worthy of inclusion in every medical library.

[1] "A Synopsis of Hygiene", by G. S. Parkinson, D.S.O., M.R.C.S., L.R.C.P., D.P.H., Lieutenant-Colonel R.A.M.C., with a section on personal hygiene by G. P. Crowden, D.Sc., M.R.C.S., M.R.C.P.; Seventh Edition; 1942. London: J. and A. Churchill Limited. Medium 8vo, pp. 720, with 16 illustrations. Price: 25s. net.

The Medical Journal of Australia

SATURDAY, AUGUST 1, 1942.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

Reference to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE WAR, QUININE AND THE MEDICAL PROFESSION IN AUSTRALIA.

THE present war is being fought with many weapons besides guns, bombs, torpedoes, land mines and other forms of explosive munitions. Among the factors with which we have to contend are deficiencies in certain weapons for the fighting of disease. Sailors, soldiers and airmen cannot give a good account of themselves unless they are maintained in robust health—alert in mind, quick and decisive in action. Medical officers of the three services—navy, army and air force—have this as their objective, but in order to attain it they must have at their disposal the necessary equipment, including drugs. In these circumstances there should be no complaint if civilians are asked to use substitute drugs—it is in the public's own interest that it should spare no effort to supply the medical branches of the armed forces with all their pharmaceutical requirements. Since medical practitioners stand between sick civilians and drugs, it is they who have to make the substitution. At the present time grave concern is being felt in Australia and other allied countries in regard to the available supplies of quinine. The position is so acute that every medical practitioner must understand the criminal folly of using quinine for any other purpose than to secure its specific action in the prevention or treatment of malaria.

Malaria has been described as the greatest killer of all diseases and the greatest single cause of disablement. Its extent is enormous. It flourishes in all tropical countries; for here the native population, with its high rate of infection, the constant heat, the moisture and water combine to make the conditions for its appearance ideal; here the *Anopheles* mosquito abounds. In most countries which enjoy a more temperate climate malaria does not appear, even though *Anopheles* may be found. Readers of this journal are well acquainted with the pathogenesis of malaria and with the remarkable life cycle of the malarial parasite and need not be reminded of it. For our present purpose, however, they may possibly need to be reminded of the tremendous toll which malaria may take of the lives of soldiers who invade a country where the disease is endemic when they come from a land in which it does not occur. R. M. Downes, in the Anne MacKenzie Oration in 1938, laid stress on this and told the story of Allenby in the Jordan Valley. Allenby was informed that if he ventured with his armies into the Jordan Valley, "the most pestilential country in the world", a loss of 100% might be expected in six months. Preventive measures with quinine made the operations of Allenby's force possible. In the same way they have been effective in this war. The point to be emphasized again and again is that malaria is a deadly scourge in the ranks of an army. The malarial parasite is in very truth "million-murdering Death", as Ronald Ross called it in a poem which he wrote in his scientific notebook on observing the completion of the life cycle of the parasite.

Quinine is the drug which cures and prevents malaria. The mythical person referred to as the man in the street knows that. Quinine has saved many millions of lives and is saving millions every day. Cinchona trees, from whose bark it is taken, are natives of the western mountains in South America, in regions extending from 10° north to 22° south latitude; and they flourish generally at a height of from five thousand to eight thousand feet above sea level. An attempt was made to cultivate them in Algeria, but without success. In 1854 the Dutch Government obtained several hundred trees from South America and planted them in Java. Some trees were obtained by Great Britain in 1859 and planted in Ceylon. Both plantings were successful, but cultivation in Ceylon was given up because of a fall in the price of quinine. Some cinchona trees are grown in India, but Java today has almost a monopoly in the production of quinine. When we remember that Java is now in the hands of the enemy, there is surely little that remains to be added. Stocks of quinine in allied countries are far from adequate, and it is unfortunately true that large supplies destined for Australia have, for reasons which need not be stated, failed to arrive. Australians have lively enough imaginations to grasp the full implications of the position. Cinchona trees cannot be planted in Ceylon, South America or anywhere else and be expected to yield quinine except after an interval of years. There are certain substitutes for quinine, and we understand that their production is to be increased by something like 8,000%; but, valuable though these substitutes are, they cannot, in the opinion of experts, entirely replace quinine. We must do what remains and husband our supplies of quinine. Quinine is used from time to time by many practitioners in obstetrical emergencies and some other non-malarial conditions in which equal results may be obtained with other remedies. Our safety as a freedom-loving people and our hope for the future demand that quinine shall be used in none other than malarial conditions; the words criminal folly already applied to its use in non-malarial conditions are not too strong. Medical practitioners are proverbially given to "shop" talk. This is the kind of "shop" that should be talked at the present time. Every practitioner who reads these words and does not discuss the subject with his or her medical neighbours will lose an opportunity of serving Australia and the democracies of the world.

Current Comment.

THE DAMAGED LIVER: NEW TESTS FOR OLD.

THE damaged liver was the subject of comment in this journal on May 6, 1939, when we reviewed some of the newer biochemical tests of liver function. We remarked then that these had not been widely accepted and employed in this country; since then one of them, the hippuric acid test, has come into fairly general use by both surgeons and physicians. Tests of liver function have been found to give useful aid to clinical judgement not only in diseases of the liver, but in thyreotoxicosis, in abdominal neoplasia and in various chronic intoxications and psychoses. It is therefore profitable to examine certain still newer tests of liver function which have been brought forward and improvements upon the old which have been developed in the last few years. Recently J. G. Mateer and others at the Henry Ford Hospital, Detroit, have made a statistical study to determine the relative sensitivity and reliability of some of these tests upon 307 patients with different types and degrees of liver disease and upon 40 normal controls.[1]

The hippuric acid test is a measure of the conjugating and detoxicating function of the liver. In this test six grammes of benzoic acid are given on an empty stomach and the urinary excretion of hippuric acid during the next four hours is estimated and the result is converted to terms of benzoic acid. Normal persons excrete the equivalent of 2·6 to 3·3 grammes of benzoic acid in the four hours, while in "surgical" types of jaundice it is found that values of less than 1·5 grammes are associated with a bad prognosis. The newer modification of this test, devised, like the original, by A. J. Quick, is called the "intravenous hippuric acid test". The subject to be tested passes urine and receives an intravenous injection of 1·77 grammes of sodium benzoate (equivalent to 1·5 grammes of benzoic acid) dissolved in 20 mils of distilled water. About five minutes are allowed for the injection. One hour later the urine is collected and its content of hippuric acid determined. A normal adult excretes the equivalent of from 0·70 to 0·95 gramme of benzoic acid during the hour period. Mateer and his associates declare that the intravenous test is reliable and much more sensitive than the original one.

The bromsulphthalein test has been modified by the introduction by D. Macdonald in 1938 of the "serial bromsulphthalein test". As in the original test, the subject receives an intravenous injection of 5·0 milligrammes of the dye per kilogram of his body weight, but instead of a colorimetric determination of the amount still left in the blood after a fixed interval being made, specimens of blood are taken every five minutes for thirty-five minutes (the first five-minute specimen being omitted), and thus the rate at which the liver removes the dye from the blood may be plotted. Macdonald reported a group of patients who had liver damage demonstrable by his serial test but not by the original method; Mateer and his associates also found the serial test by far the more sensitive.

Another test of liver function introduced in recent times is the cephalin-cholesterol flocculation test described by F. M. Hanger in 1938-1939. Hanger observed that the serum of persons suffering from hepatic damage is often capable of flocculating a colloidal suspension of a cephalin-cholesterol emulsion while that of persons with normal livers seldom or never does so. Mateer and his associates found this test good and sensitive and recommended it for routine use.

It is noteworthy that the newer modifications subject the liver to a shorter, sharper test than did the original tests; an abnormal liver may perform the same amount of work as a normal one, if allowed sufficient time, but a normal liver can perform a stipulated task more rapidly than an impaired liver.

There is no doubt that finality has not yet been reached in the interpretation of tests of liver function. Mateer and his associates refer to the "dissociation" of these tests—that is to say, the difference in result between one test and another performed on the same patient at the same time—and they state that it is necessary to conduct "at least several" different tests if one expects to demonstrate impairment of liver function in the highest possible proportion of patients having such impairment. Apart from this, tests of this or that function of the liver may not give a clear idea of the state of the organ as a whole. It is only a few years since apparent impairment of galactose tolerance in thyreotoxicosis was attributed to hepatic damage, although it was soon realized that a contributory factor to the abnormal results of the galactose tolerance test in this disease was an abnormally rapid absorption of sugars from the alimentary canal. Again, it is well known that serious depression of liver function may be present one day and not the next; it may pass off like a bad mood, and presumably may do so at the same time. Hence a too gloomy view of the hepatic workings should not be taken on the strength of a single test. Although much has been written of tests of liver function made in the presence of serious hepatic and other diseases, little or nothing has been said of their correlation with those symptoms which are usually taken to indicate that the liver is at fault, or of the liver function in many minor illnesses, such as influenza, where these symptoms may be very pronounced. Investigation of these matters will no doubt be forthcoming in due time and will be of considerable interest.

[1] *The American Journal of Digestive Diseases*, January, 1942.

DIETARY HEPATIC INJURY IN RATS.

THE vitamin *B* complex is an ever-recurring theme in medical literature today and the reason is not far to seek; for as vitamin *B* vanishes from our food, so it must appear in our medicine. It seems to be abundant in the cruder foods, in wholemeal bread, in yeasts, in home-brewed beer and the toddies and other concoctions of native races, but it tends to be scarce in a civilized diet. One is reminded of the work of George Bray who vanquished most of the maternal and infant mortality among the natives in Nauru by restoring to their ration of white flour and sugar the palm-juice toddy forbidden by the Government. Now yet another role for the vitamin *B* complex appears; it looks as though it may be concerned in the prevention of cirrhosis of the liver.

Various observers have reported the occurrence of cirrhosis of the liver in rabbits and its prevention by the administration of yeast. In 1939 Paul György and Harry Goldblatt observed a similar phenomenon in rats. Hepatic injury, mainly in the form of acute focal or diffuse necrosis combined with fatty infiltration, occurred irregularly in young rats fed on diets deficient in vitamin *B*, but supplemented with thiamin, riboflavin and pyridoxin. Injury to the liver was never encountered in rats fed on a diet exactly similar except that yeast was substituted for the pyridoxin. Nor was hepatic injury found in investigation of riboflavin deficiency when 500 rats were fed the same basal diet supplemented with thiamin and yeast extract. György and Goldblatt[1] now publish experiments designed to elucidate the cause of this dietary injury to the liver. One common denominator emerged from their earlier observations. This was the possibility of low food intake with, at the same time, a sufficient supply of several, at least, of the known members of the vitamin *B* complex. In the diets used for rats, a low food intake meant a low casein supply and therefore a diminution of the so-called lipotropic activity of casein. For this reason the authors employed various rations low in casein and moderately high in fat. Furthermore, since the lipotropic action of casein is generally considered to be completely determined by the action of cystine,

[1] *The Journal of Experimental Medicine*, April 1, 1942.

methionine and choline, supplements of one or more of these substances were added to the diets used. To complete the argument, it should be mentioned that choline and methionine are present in yeast and that in some ways these substances are antagonistic to the amino-acid, cystine.

Rats maintained on a diet low in casein with a moderately high or a high content of fat and without choline, readily exhibited hepatic injury after 100 to 150 days. The addition of l-cystine had an aggravating effect on the production of cirrhosis of the liver, whereas a supplement of choline alone reduced the severity and the incidence of hepatic injury, although not decisively. The combined administration of l-cystine with choline or of d-l-methionine in adequate doses, however, proved to be highly effective in preventing injury to the liver. The changes shown varied from diffuse necrosis resembling human acute or subacute yellow atrophy to advanced portal cirrhosis. Changes in the kidneys, described by György and Goldblatt as "diffuse necrotizing nephrosis", were a frequent accompaniment of the hepatic injury. This is a very interesting finding, especially when the authors add that in previous experiments, in which much younger rats were fed on a diet deficient in choline, the kidneys showed changes resembling human bilateral symmetrical cortical necrosis.

One is tempted to jump to conclusions *per analogiam*, but, of course, application of the results of these experiments to conditions in human beings is little more than speculation. Nevertheless it is interesting to remember that similar ætiological conditions are present in human alcoholic cirrhosis. A low intake of protein combined with a variable supply of the vitamin *B* complex (including choline) is a feature of the diet of persons addicted to alcohol. György and Goldblatt also venture to call attention to the identical nutritional ætiology of hæmorrhagic cortical necrosis of the kidneys and of necrosis of the liver, and to the occurrence of both these lesions in pregnant women. Their experiments are being continued, to try to find out whether or not cirrhosis of the liver once produced can be influenced therapeutically. The results will be awaited with great interest.

TREATMENT OF THE MENOPAUSAL SYNDROME.

THE efficiency of many œstrogenic compounds has been well established, but the present standard method of administration is far from satisfactory as it requires frequent hypodermic or intramuscular injections of the hormone preparation, usually in oil, and repeated as often as once to three times a week. This has been pointed out by H. G. Bennett and Richard W. Te Linde,[1] who have recently reviewed the literature dealing with the subcutaneous implantation of œstrogenic compounds and have reported their results with this method of treatment in patients suffering from what they call the menopausal syndrome. The subcutaneous implantation of drugs is not a new idea, for many reports have appeared of the favourable results obtained by the subcutaneous implantation of tablets of synthetic adrenal cortical hormone (desoxycorticosterone acetate) in patients with adrenal insufficiency; and in many clinics this has become a firmly established method of treatment.

Following the implantation of a standard routine dose of 50 milligrammes of theelin, Bennett and Linde found that improvement generally began within two weeks of implantation and lasted on an average for sixteen weeks. The relief from hot flushes was taken as the criterion of improvement; but many of the patients considered the results satisfactory even though the hot flushes did not absolutely disappear. The freedom from unpleasant side effects such as nausea and vomiting, breast hypertrophy and chronic cystic mastitis which sometimes accompany other methods of ovarian replacement therapy was a pleasing feature of their results. The relief from the menopausal symptoms was just as satisfactory with the implantation of diethylstilbœstrol as with theelin, but with the former compound there were often disagreeable side effects in the form of nausea and abnormal uterine bleeding, and therefore this former drug is not recommended for implantation. These authors found that the best results were obtained in those women in the acute phase of the menopause, whether physiological, surgical or post-irradiation, rather than in the chronic phase; and they accordingly suggested that treatment should be prompt. In the long-standing cases patients often develop a neurosis and in Bennett and Linde's series only 60% of the patients in whom symptoms had been present for more than one or two years admitted to relief after therapy. That the symptoms in these patients were chiefly neurotic in origin was suggested by the fact that small repeated doses of phenobarbital were nearly as effective as the œstrogen therapy. From a study of vaginal smears in their patients Bennett and Linde concluded that the smears were not a dependable indicator of the necessity for the œstrogen or of its effectiveness in the treatment of symptoms. Similarly laboratory methods of estimation of the follicle stimulating hormone in urine may be useful in research, but they are probably unnecessary in the treatment of the individual patient.

As a result of the implantation of diethylstilbœstrol there is often a benign endometrial proliferation which may give rise to bleeding at or after the menopause, and it may be difficult to be certain whether this bleeding is due to the endocrine treatment or whether it is dependent on some serious lesion such as carcinoma of the body of the uterus. A diagnostic curettage may be necessary to decide the point. It is therefore important to use an œstrogenic hormone such as theelin which does not cause bleeding. Many of the œstrogenic substances have a carcinogenic action in laboratory animals and it has been shown that the injection into guinea-pigs of large amounts of these substances will regularly produce uterine fibromata. Fortunately, this probably has no clinical bearing when theelin is used, for this substance is the weakest growth stimulator of the various œstrogens. Nevertheless this is a point which should receive further consideration.

The high incidence of effective results (93·4% in Bennett and Linde's series) and the prolonged action of implanted theelin without untoward side effects suggest that this method of treatment is worthy of trial in patients with menopausal symptoms of recent origin.

[1] *The Journal of the American Medical Association*, April 18, 1942.

THE CENTENARY OF THE FIRST USE OF ETHER AS AN ANÆSTHETIC.

NOTICE should be taken of the fact that in this year medicine celebrates the centenary of the first use of ether as an anæsthetic. To mark this event A. D. Marston dealt with the centenary in his President's address to the Section of Anæsthesia of the Royal Society of Medicine last November.[1] Marston tells how the use of ether as an anæsthetic arose from its use at parties as an alternative to nitrous oxide gas in the production of "frolics". If no pain was felt during "frolics" when the participant tumbled and reeled, why should not the business be pushed further? Thus argued Crawford Williamson Long, a young medical man who gave parties at his own house. He found nitrous oxide hard to get and obtained ether as a substitute from a local dentist. Thus it happened that on March 30, 1842, Long gave ether to a young man named James Venable and removed a cyst from his occipito-cervical region. Marston quotes from the writings of Jim Vandier, a friend of Long, an account of the memorable event. A crowd had gathered, determined to lynch Long if the boy failed to survive. He did survive, and Marston's account of the later development of the use of ether should be read by those who do not know the history. Had it not been for the war we should probably have heard a great deal more of this important centenary.

[1] *Proceedings of the Royal Society of Medicine*, February, 1942.

Abstracts from Medical Literature.

THERAPEUTICS.

Agranulocytosis.

R. OTTENBERG (*The Journal of the Mount Sinai Hospital, New York*, January-February, 1942) describes a new treatment for leucopenic states. Nucleotide therapy has not been generally accepted; transfusion has a limited effect. The injection of foreign protein into the donor, however, produces a temporary leucocytosis of 15,000 to 30,000 per cubic millimetre. Injection of foreign protein into the patient suffering from granulopenia generally fails to produce a leucocytosis because of the damaged bone marrow. In the case recorded Ottenberg injected 15 million killed typhoid bacilli intravenously into the donor of the blood the day before he utilized the blood for transfusion. The result was that on the day of the transfusion the donor's blood contained 28,500 leucocytes per cubic millimetre. The patient, who had a sore throat, was given 15 grains of sulphonamide in twelve hours; her condition improved, but a week later she developed high fever, *otitis media*, pharyngitis and impetigo. A blood count revealed 9,100 leucocytes per cubic millimetre with only 0·5% polymorphonuclear cells. A transfusion of 150 cubic centimetres from the donor mentioned above led to the immediate remarkable improvement. The patient, who had been very seriously ill, felt better at once, the temperature fell from 105° to 101° F. and then to normal in a few hours. The blood count a day later revealed 34% granulocytes among 10,000 leucocytes. Complete recovery occurred.

Gout.

C. MCEWEN (*Journal of the Mount Sinai Hospital, New York*, January-February, 1942) describes the use of diets with a high fat and high purine content in the diagnosis of gout. Gouty patients were given a diet containing high fat content (260 grammes) with low protein (45 grammes) and carbohydrate (50 grammes) content. The protein was mainly derived from milk, eggs and cheese. With this diet blood uric acid content rose to a slightly higher level than it had been before, and patients suffered pains in the great toe, feet and other joints typical of gout. Other patients were given diets with a high carbohydrate content (300 grammes) with moderate protein (70 grammes) and fat (85 grammes) with resultant improvement in symptoms and lowering of the blood uric acid level. The blood uric acid content was between 5 and 9 milligrammes *per centum* during attacks of gout; it rose to its maximum, 11 milligrammes in one case, after ingestion of a diet with a high purine content. The purine-rich diet contained 119 grammes of carbohydrate, 143 grammes of protein and 87 grammes of fat. Meat was given at every meal, and thymus, liver or sweetbreads once a day. With this diet gouty symptoms occurred more readily and the blood uric acid level was higher than with diets rich in fat or in carbohydrates. There appeared to be no doubt in these experiments that a diet with a high carbohydrate content with 70 to 80 grammes each of fat and protein was by far the most satisfactory both from the clinical and biochemical aspects of gout.

Insulin.

C. C. BAILEY AND A. MARBLE (*The Journal of the American Medical Association*, February 28, 1942) discuss the action of histone zinc insulin, globin zinc insulin, and clear protamine zinc insulin. The turbid protamine zinc insulin at present used is effective, but has certain disadvantages. Histone insulin is prepared by adding to insulin the simple protein histone, derived from the thymus. It has a prolonged action. One dose before breakfast begins to act within two hours and continues to act throughout twenty-four hours. There are transient rises of blood sugar during the day, but this insulin has much the same action as turbid protamine zinc insulin, except that it begins to act much earlier, and causes fewer hypoglycæmic reactions. Its only disadvantage is that it tends to clump and to adhere to the neck of the phial. Globin zinc insulin is a long-acting insulin which has less initial effect (in the first two hours) than histone zinc insulin, and a slightly less prolonged action than turbid protamine zinc insulin. It tends to cause hypoglycæmia between 2 and 5 o'clock p.m. and for this reason it is advisable to give carbohydrate in the mid afternoon. It is best not to give carbohydrate at bedtime with this insulin in order to avoid hyperglycæmia at night. With histone zinc insulin, on the other hand, it is advisable to give a feed at bedtime, as with turbid protamine zinc insulin. Clear protamine zinc insulin is long acting, lasting about eighteen to twenty-four hours, but it is less effective in controlling the blood sugar than other insulin, and is more likely to give rise to hypoglycæmic reactions. The authors investigated a series of diabetic patients in their reaction to the insulin mentioned above, and they concluded that ordinary insulin for its rapid effect and turbid protamine zinc insulin for its prolonged effect, are at present the most satisfactory forms of insulin for general use.

The Nephrotic Crisis.

K. EMERSON AND D. D. VAN SLYKE (*Journal of the Mount Sinai Hospital, New York*, January-February, 1942) describe the symptoms of the nephrotic crisis and its treatment by intravenous injection of amino acids. The nephrotic crisis is a recurrent febrile attack with symptoms of peritonitis in a patient suffering from nephrosis. The onset is sudden with a temperature up to 106° F. in three or four hours, chills, generalized abdominal pain and rigidity, nausea, vomiting and prostration. A leucocytosis occurs and a pneumococcus, streptococcus or other organism is often found in cultures of peritoneal fluid. Bacteriæmia and death or recovery may occur. Farr showed that a marked loss of urinary non-protein nitrogen preceded the crisis. The normal α-amino acid nitrogen of the blood plasma (3·5 to 5·0 milligrammes per 100 cubic centimetres) was consistently decreased in the nephrotic syndrome, to 2·5 to 3·5 milligrammes per 100 cubic centimetres. During the acute nephrotic crisis the figure dropped to below 2·5 milligrammes. This was the critical level. Recovery was associated with an abrupt rise in the amino acid in the plasma. During the crisis gastro-intestinal symptoms are so acute that feeding is impossible. Consequently the amino acids have been given intravenously in the form of casein hydrolysate. It is not claimed that this treatment has prevented or shortened the nephrotic crisis, but the fact is that since the introduction of amino acid therapy the mortality from crises has diminished remarkably. MacLeod and Farr observed that nephrotic children carry in their throats for a long time the organisms which subsequently invade the blood stream in a nephrotic crisis.

NEUROLOGY AND PSYCHIATRY.

Folie à Deux.

ALEXANDER GRALNICK (*The Psychiatric Quarterly*, April, 1942) presents a review of 103 cases of *folie à deux*. He notes the prominence of sister combinations as compared with the relatively fewer brother combinations. Next in order of frequency are the mother-daughter cases. He finds the role of homosexuality difficult to assess. He believes that cultural factors play a part in the ætiology, making a woman more suggestible and dependent upon a dominant woman. He thinks that sisters are often involved because they are frequently mother substitutes. In the majority of cases the older person is first affected; the younger person is usually dependent and submissive. Most patients harbour persecutory delusions and display the paranoid reaction type. The writer found hallucinations absent in about half the cases. He distinguishes between the "imposed" and "communicated" types, the former tending to recovery within six months.

Juvenile Amaurotic Idiocy.

HAVING drawn attention to the four types of amaurotic idiocy, George A. Jervis, Leon Roizin and William H. English (*The Psychiatric Quarterly*, January, 1942) present a case of this juvenile type and contribute to the discussion of its relationship to disorders of fat metabolism. Clinical evidence and autopsy findings are recorded in detail; and the authors refer to theories that this disease is characterized by a disturbance of fat metabolism in which it is thought that amaurotic idiocy represents a type of protagon lipoidosis localized in the central nervous system. The analogy to Gaucher's disease is mentioned. The faulty metabolism results in the storage of unmetabolized products owing to lack of specific enzymes which at this stage are not known.

The Incidence of Psychoses and Other Mental Abnormalities in the Families of Recovered and Deteriorated Schizophrenic Patients.

OTTO KANT (*The Psychiatric Quarterly*, January, 1942) has studied the familial abnormalities in 50 recovered and a similar number of deteriorated schizophrenic patients. The incidence of familial functional psychoses was 44% in the recovered group and 36% in the deteriorated group. The highest

incidence of familial schizophrenia was found among the deteriorated catatonic group, while in the recovered group no familial schizophrenia was found among the catatonics. Manic-depressive psychosis of a familial type was found to be five and a half times more frequent than actual schizophrenia in the recovered group. These studies confirmed the accepted idea that manic-depressive insanity was more dominant in the direct line and schizophrenia more frequent in the collateral branches of the family tree.

"Metrazol" Therapy in Affective Psychoses.

EUGENE ZISKIND, ESTHER SOMERFIELD-ZISKIND AND LOUIS ZISKIND (*The Journal of Nervous and Mental Disease*, April, 1942) have investigated the effect of "Metrazol" in the treatment of 43 cases and have compared these results with those from 47 patients who remained untreated. All patients were suffering from affective psychoses. The authors claim that 92% of treated patients achieved remission as against 72% of untreated patients. Full remission was gained by 82% and 38% respectively. The average duration of the illness was one and a half months, compared with six months in the case of untreated patients. The writers consider that failure to effect a remission is frequently due to inadequacy of treatment which should consist of an active phase of injections until the symptoms disappear, a reinforcement phase of a few further injections and an additional number of injections, should symptoms reappear. Fear of the injection constitutes the chief barrier to the treatment. Failure to produce a convulsion may make the patient worse and cause increased fear. Memory impairment is sometimes a marked feature of the treatment, but it is always temporary. Contraindication on the grounds of physical disability is liable to be over-emphasized. The number of accidents is very small. Spinal fractures which cause no clinical symptoms are relatively common and are of no more moment than they are in epileptics. The writers believe that shock treatment is a quantitative procedure and they state that convulsions induced at the rate of more than two a week are likely to produce memory defects; but they prefer to leave the question open as to whether "Metrazol" therapy can produce definite and permanent brain damage.

The Family in a World War.

AMONG the factors which make for group solidarity, George E. Gardener (*Mental Hygiene*, January, 1942) finds the following three fundamental factors: security, participation in joint enterprises, and the presence of strong parental figures. Without economic security, in the form of employment, production and consumption of goods, no amount of psychiatric treatment is of value; but in the absence of this security, that security which is based on emotionally satisfying parental-child relationships will go far to maintain family stability and prevent the delinquencies, neuroses and other deep-seated emotional conflicts which condition abnormal behaviour. Indeed in a state of war this emotional security is essential. The war, of course, gives ample opportunity for stability within the family unit through participation in a common cause. No one is quicker than a child to note the inconsistencies and prejudices of parents; but war gives opportunities for leadership so that the children may incorporate in themselves the standards and ideals exemplified in the parents.

Akinetic Mutism with an Epidermoid Cyst of the Third Ventricle.

THE case of a girl of fourteen with an epidermoid cyst of the third ventricle is reported by H. Cairns, R. C. Oldfield, J. B. Pennybacker and D. Whitteridge (*Brain*, Volume LXIV, Part 4, 1941). This lesion produced mutism, loss of voluntary and emotional movement with the exception of movement of the eyeballs, apparent loss of emotional feeling and other symptoms. On three occasions the cyst was aspirated and the symptoms disappeared almost immediately. Finally the cyst was removed and eight months after the operation the child was still free from headache, mutism, drowsiness and polyuria and was able to do routine work. The writers believe that the akinetic mutism may be interpreted as due for the most part to a disturbance of cortical function and not to any increased intracranial pressure. They think the cortex is largely put out of action by local pressure at the diencephalic level.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 195, of July 16, 1942.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointments.—Edward Winston Freshney is appointed Surgeon Lieutenant, dated 25th May, 1942; John Henry Begg, Arthur Noel Berkeley Fitzhardinge and Colin Keith Hambly are appointed Surgeon Lieutenants, dated 9th June, 1942.

Transfer to Retired List.—Surgeon Lieutenant Robert Philip Booth is transferred to the Retired List, dated 23rd June, 1942.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

Captain VX67344 R. V. Pratt is transferred from Reinforcements, with regimental seniority in accordance with his Army seniority, 9th April, 1942.

Captain VX56445 J. Morlet is transferred, with regimental seniority in accordance with his Army seniority, and to be Major (temporarily), 12th May, 1942.

To be Major (temporarily).—Captain VX48253 C. P. Manson, 12th May, 1942.

To be Major (temporarily).—Captain S. A. McDonnell, 12th December, 1941.—(Ex. Min. No. 161—Approved 15th July, 1942.)

Major W. W. Lempriere is appointed to command a Field Ambulance, 22nd December, 1941.

To be Lieutenant-Colonel (temporarily).—Major W. W. Lempriere, 22nd December, 1941.

To be Majors (temporarily).—Captains R. F. A. Becke and H. W. Anderson, 22nd December, 1941.

Captain (Temporary Major) R. Drummond, M.B.E., is transferred and to be Lieutenant-Colonel (temporarily), 14th April, 1942.

Captain B. G. G. Fegent is transferred, 20th April, 1942.

Australian Efficiency Decoration.—Lieutenant-Colonel NX70413 N. D. Barton, Australian Army Medical Corps.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

Captain (Temporary Major) M. G. Edison is transferred, is appointed to command a field Ambulance and to be Lieutenant-Colonel (temporarily), 1st May, 1942.

Major (provisionally) Q119947 E. D. Ahern, E.D., is transferred and to be Lieutenant-Colonel (temporarily), 3rd June, 1942.

Lieutenant-Colonel D. L. Barlow, M.C., E.D., relinquishes command of a Convalescent Depot and is transferred to the Reserve of Officers (A.A.M.C.), 23rd September, 1941.

NEWS.

WE are indebted to Surgeon Captain W. J. Carr, Director of Naval Medical Services, for the following report on the work of a young sick berth attendant of the Royal Australian Navy Sick Berth Branch. The report is from the commanding officer of the vessel, and, as Surgeon Captain Carr remarks, illustrates the value of intelligent and well-trained personnel in ships in which medical officers are not available.

On February 19, 1942, H.M.A.S. "——" was in Darwin when the enemy made a heavy attack on the port.

Sick Berth Attendant "X.Y.Z." was in a nearby ship undergoing dental treatment, but he took the first opportunity of rejoining his ship, which sailed that night under orders to pick up survivors from ships sunk at sea.

The following morning the task of picking up survivors from the "X.X." was begun. These people were spread over several miles of an exposed beach on an island, and some were on top of a low cliff further south. Before reaching shore, they had been ten hours in the water, drifting in lifebelts, so most were in a state of utter exhaustion, a further twenty-four hours without food or water having proved fatal to some of them already. It was only owing to the skill and quick perception of our sick berth attendant that so many wounded survived the journey through the surf to the ship and thence back to Darwin.

He organized stretcher parties, saw to the landing of water and first aid requirements, and kept up a constant flow of instructions to his willing but unskilled helpers. Officers and crew were so largely occupied in managing the ship and boat in the surf that, to a great extent, the actual transport of the wounded was left entirely to the sick berth attendant's ingenuity.

For such cases as men so badly burnt that it was impossible to touch them without coming into contact with burnt flesh, he devised means of getting them to the ship. Once all the survivors were aboard, the task having taken six hours, he tirelessly worked among the wounded, giving injections, dressing burns and bullet wounds.

The surgeon who received the cases in Darwin gave him the highest praise, and it is solely to his credit that only two cases died out of a large number of badly wounded. He had each man labelled for reference at the hospital and, before making port, he gave me information to be signalled that considerably helped the medical staff ashore in their arrangements for the disposal of the wounded. It was about 4 a.m. before these were landed, and there were more to pick up yet.

As the ship's stock of medical supplies was exhausted, he lost no time in finding and acquiring more. The hospital had been bombed, and most shipping was at sea, but by midday he was back with various drugs and dressings and a couple of spare stretchers, having used his initiative in obtaining these. The ship proceeded to sea to pick up more survivors, and the sick berth attendant, in the light of the previous day's experience, started to lay out part of the mess decks as a hospital. Later, when he could have rested, two of the ship's company demanded his attention, showing signs of shell shock.

At dawn he was away in the boats again, undertaking a long passage through reefs and surf. With the same skill that he had shown on the previous occasion, he organized the transfer of the wounded survivors of the "Y.Y.", in a much greater state of exhaustion than those of the "X.X.", and also the wounded from an aircraft which had been shot down. As he was approaching the ship with a boatload of wounded, the ship was attacked by an enemy bomber, and he used his efforts of persuasion on some of the terrified and shell-shocked wounded who tried to jump overboard. These men undoubtedly owe their lives to his skill and tireless care of them until they were finally landed.

CASUALTIES.

ACCORDING to the casualty list received on July 22, 1942, Major G. F. S. Davies, A.A.M.C., Camperdown, New South Wales, and Major B. L. W. Clarke, A.A.M.C., Ascot, Queensland, are reported to be missing abroad.

According to the casualty list received on July 23, 1942, Captain F. J. Cahill, A.A.M.C., Flemington, Victoria, Lieutenant-Colonel A. E. Coates, A.A.M.C., Toorak, Victoria, Captain V. A. Conlon, A.A.M.C., Lockhart, New South Wales, Major T. P. Crankshaw, A.A.M.C., Malvern, Victoria, and Lieutenant-Colonel H. F. Summons, A.A.M.C., Kew, Victoria, are reported to be missing overseas.

According to the casualty list received on July 23, 1942, Captain R. W. Greville, A.A.M.C., Hawthorn, Victoria, is reported to be missing overseas.

According to the casualty list received on July 24, 1942, Major E. A. Marsden, A.A.M.C., Camperdown, New South Wales, is reported to be missing abroad.

According to the casualty list received on July 27, 1942, Captain F. H. Mills, A.A.M.C., Bellingen, New South Wales, Captain R. M. Mills, A.A.M.C., Narrabeen, New South Wales, Lieutenant-Colonel W. C. B. Harvey, A.A.M.C., Sydney, New South Wales, Captain J. P. Catchlove, A.A.M.C., Elwood, Victoria, and Captain J. S. S. Winter, A.A.M.C., Toorak, Victoria, are reported to be missing overseas.

According to newspaper reports, Dr. Charles A. Tunstall, of the Merchant Navy, was killed when the steamer *Macdhui* was lost as a result of enemy action at Port Moresby in June, 1942.

Correspondence.

RADIOLOGICAL SURVEY OF CHILDREN'S CHESTS FOR TUBERCULOSIS.

SIR: It has recently been announced in the Press that a radiological survey of children's chests for tuberculosis is to be made. I view such survey with great apprehension, especially in the case of young children.

A feeling is becoming engendered in the public mind of the infallibility of the X-ray diagnosis in pulmonary tuberculosis; that the X-ray alone can say "this is tubercular, this is not tubercular".

I have heard it said as a considered opinion by a number of physicians that radiologists have at times diagnosed, as tubercular, children whom clinical signs, family history, Mantoux test and ultimate progress did not in any way suggest were suffering from this disease. Several of the physicians at the Royal Alexandra Hospital for Children have stated that from their experience both of the in-patient and out-patient department, pulmonary disease of tuberculous origin is exceedingly rare in children up to the age of thirteen years.

Many years ago I gathered the opinion that the percentage of positive cases reported in chests of young children varied greatly with the individual radiologist, and my opinion still remains the same. This can mean only one of two things, either that cases are labelled erroneously as tubercular, to the great mental anguish of the parents, or that cases are being "missed" by the reporting radiologist.

It would be interesting and valuable to obtain the opinion of the physicians at the Royal Alexandra Hospital for Children as to the incidence of pulmonary tuberculosis among children at this hospital, and in its out-patients, for they are the people who should have the greatest knowledge of this particular subject. Most radiologists, I think, will agree with me when I say that the diagnosis of diseases in the children's chest is at times far from easy.

Yours, etc.,

H. R. SEAR,
Honorary Radiologist, Royal Alexandra Hospital for Children.

Craignish,
185, Macquarie Street,
Sydney.
July 9, 1942.

DOCTORS AND THE DRINK TRAFFIC.

SIR: I would like to express my agreement with Dr. Nye's letter on drunkenness. Undoubtedly the too persistent drinker is a case for the psychiatrist. If the Army adopted the plan of sending him for a mental examination, it would go far to "debunk" the idea that over-drinking is a symbol of good fellowship. Actually it symbolizes insufficient cortical control.

The importance of this problem cannot be overstressed. Over-drinking is a symptom of a pathological state. It may be argued that a contributory factor is our failure, even now, to realize our national peril. The over-drinker should be regarded as "scabbing" on the community and letting his mates down on active service. Such an attitude would assist in "deglamourizing the drunk".

In fairness to the soldiers, it must be conceded that our prophylaxis needs overhauling. Dr. Nye rightly stresses the need for a reorganization of the bar facilities so that people are encouraged to sit around a table as is done on the Continent. Here are three more needs: (*a*) Drastic diminution of the alcoholic content of all alcoholic beverages so that the drinking strain is renal rather than cerebral. The present content is far too high. Many men are said to become inebriated on two beers. (*b*) Soldiers should be permitted to drink for a period after the bar has closed, but not in the bar. The psychological effect of having to finish drinking at an early hour is to create rush drinking. Chain drinks give no time for warning. The stomach is loaded to excess before the mind is aware of its approaching narcosis. (*c*) "Shouting" drinks should be a punishable offence for the duration of the war and a year after. Every soldier and civilian should buy his own drink. "Shouting" drink is a psychological gesture based on the self-regarding instinct and serves no useful purpose beyond contributing to the publican's economic welfare. We do not give daily presents of clothes. It is a simple extension of the principle to extend it to beer.

As a profession I feel that we ought to take a greater interest in sociology and within this category the problem of alcohol shows a pressing need for urgent action.

Yours, etc.,

JOHN BOSTOCK.

Brisbane Clinic,
Wickham Terrace,
Brisbane.
July 15, 1942.

ANÆSTHESIA AND DENTAL SURGERY.

SIR: I would like to express my appreciation of your leading article in THE MEDICAL JOURNAL OF AUSTRALIA of June 27, 1942, headed "Anæsthesia and Dental Surgery".

Your appeal to the medical profession to assist the dentist by providing a suitable and safe type of anæsthesia will doubtless find a willing response. Open ether is a most undesirable anæsthetic for multiple dental extractions, especially where the teeth contain a number of old and fragile metal fillings, and, in my judgement, should never be given. The suggestions of the Victorian Branch of the Australian Dental Association may go some little way towards preventing the catastrophe of inhalation of blood or fragments of teeth or fillings, but are far from ideal, as I am sure Mr. George Finlay and those with him would readily accord.

Endotracheal ether capably administered leaves nothing to be desired from the point of view of safety for the patient and freedom from anxiety on the part of the operator, and I would like to see this made the standard method of anæsthesia for any dental or oral operation. Of course, I am not overlooking the refinement of endotracheal nitrous oxide and oxygen or cyclopropane, but am referring to the ordinary anæsthetic for the ordinary dental patient where general anæsthesia is indicated.

Permit me now to pay tribute to the skill and adaptability of the great majority of medical practitioners of this city with whom I have been associated for a number of years. The fact that so many are able and willing to give an endotracheal anæsthetic which is safe and satisfying is a matter that reflects considerable credit upon the teaching in our medical school and training hospitals, and I am glad to pay this tribute not only to their skill, but also to their willing cooperation and consideration in the matter of fees. Of practitioners outside of Sydney, I am unable to speak from experience, but am sure that at least in the larger centres there are many who are skilled in this service.

No dentist in this city has to go far to find an anæsthetist who can provide him with an anæsthetic that will free him from care and smooth his way. The dentist who benefits from this skilled service should, wherever possible, see that the patient is prepared to pay an adequate fee, and, if the matter is rightly represented beforehand, most patients readily respond. Where, through force of circumstances, this is not possible, I have always found our medical colleagues most willing to cooperate.

Yours, etc.,

University of Sydney,
July 16, 1942.

E. STANLEY WALLACE.

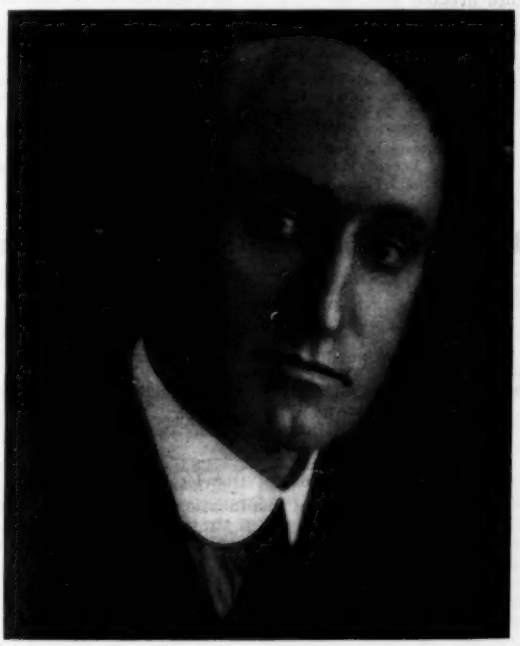
Obituary.

ARTHUR AUBREY PALMER.

DR. ARTHER AUBREY PALMER, whose death was recorded recently in these pages, held a high and honourable place in Australian medicine. He was known to more than one generation of students at the University of Sydney Medical School and he was recognized as an authority on medical jurisprudence. By the depth of his knowledge, his sincerity, his transparent honesty and his strength of character, he earned the admiration, the confidence and the respect of the medical profession, of the legal profession and of the police force of New South Wales. There are few who have wielded a wider influence by their professional work.

Arthur Palmer was the son of George Palmer, a pastoralist, of Conadgery Station, Bogan River. He was born on March 27, 1871. He went to school at All Saint's College, Bathurst, and went to Edinburgh to study medicine. He was a successful student and obtained the degrees of Bachelor of Medicine and Master of Surgery of the University of Edinburgh; later he became a Fellow of the Royal College of Surgeons of Edinburgh. He also became a member of the Royal College of Surgeons of England and a licentiate of the Royal College of Physicians of London. During his student days at Edinburgh he gained the gold medal in anatomy, as well as two junior medals in the same subject. Not unnaturally he became a demonstrator in anatomy. First of all he was appointed junior demonstrator under Professor J. Symington. When Professor Symington took the chair of anatomy at Queen's College, Belfast, Palmer became his principal demonstrator, and later on he returned to Edinburgh as demonstrator at Surgeon's Hall.

Arthur Palmer came to Sydney in 1899. He soon became associated with the department of anatomy at the University of Sydney and for many years acted as examiner in that subject. Though he was searching as an examiner he was always fair to the student—he tried to discover what the student knew and whether the knowledge was well grounded and few students were not at their ease with him. In 1909 he was appointed police surgeon at Sydney. He was always fair and just in his treatment of members of the force and he earned their respect and affection. In the latter years of his appointment it was interesting to take a walk with Palmer through some of the streets of Sydney and to see the way in which he was greeted by every policeman on duty. When he retired from the position of police surgeon



the members of the force in recognition of his sterling qualities presented him with a cheque which was intended, and actually was used, to give him and his wife a trip to Great Britain and Europe. For many years in his capacity of government medical officer he conducted post-mortem examinations for the Sydney coroner. Here again the soundness of his decisions was shown by the respect with which his evidence was always received by members of the legal profession. Of his relations with the British Medical Association much might be written. He was a prominent member of the New South Wales Branch and served on its council for many years. He was President in the year 1918-1919. His opinions always carried weight on account of the obvious sincerity with which he expressed them. Eventually in 1939 the Branch Council conferred on him the highest honour at its disposal by electing him a Vice-President. In 1929 Arthur Palmer shared with Dr. T. W. Lipscomb the onerous duties of acting as Honorary Secretary of the Third Session of the Australasian Medical Congress (British Medical Association) held in Sydney under the presidency of Dr. George Abbott. This congress is remembered as a triumph of organization. Palmer also took a lively interest in the welfare of the Royal Alexandra Hospital for Children, being a member of its board for many years and latterly a Vice-President. As lecturer in medical ethics and in medical jurisprudence at the University of Sydney he had the opportunity to influence many hundreds of students and it may be stated without hesitation that his influence was invariably for good; it could not be otherwise. He was a member of the Dental Board of New South Wales.

Arthur Palmer lived a full life and one rich in achievement; he did in truth leave the world a better place for his having lived in it. Much sympathy has been expressed for his wife who survives him.

NOTICE.

THE next meeting of the clinical section of the medical board of the Royal Prince Alfred Hospital will be held on August 13, 1942, in the A2 lecture theatre at the hospital. The meeting will start at 4.30 o'clock p.m., when demonstrations will be given by Dr. H. H. Schlink and Dr. A. Walker-Smith. All medical practitioners are invited to attend.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Darvall, Anthony Roger, M.B., B.S., 1942 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Ferguson, David Alexander, M.B., B.S., 1942 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Cohen, Douglas Harry, M.B., B.S., 1942 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Browne, Gordon William, M.B., B.S., 1942 (Univ. Sydney), Newcastle General Hospital, Newcastle.

Sutton, David Harvey, M.B., B.S., 1942 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Tomb, John Walker, M.B., B.Ch., 1910, D.S.M., 1911, M.D., 1912 (Univ. Dublin), Bank of New South Wales, Sydney.

Brenner, Maksmiljan, M.B., B.S., 1942 (Univ. Queensland), Mount Morgan District Hospital, Queensland.

The following additional qualification has been registered:

Kenny, Patrick John (M.B., B.S., 1936, Univ. Sydney), F.R.C.S., 1940, England, Australian Imperial Force.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 to 1940, of Queensland, as duly qualified medical practitioners:

James, Clive Henry Reynolds, M.B., B.S., 1923 (Univ. Melbourne), Hughenden.

Valese, Peter, M.B., B.S., 1942 (Univ. Sydney), General Hospital, Toowoomba.

Medical Appointments.

Dr. Philip Lawrence Kenneth Addison has been appointed a Member of the Railway Service Superannuation Board, New South Wales, until October 5, 1945, pursuant to the provisions of section 110 of the *Government Railways Act*, 1912 *to* 1934.

Dr. Hugh Gilmour Wallace has been appointed a member of the Dental Board for a period of three years from July 1, 1942, in pursuance of the provisions of the *Dentists Act*, 1934, of New South Wales.

Books Received.

"Monographs from the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne; Number Four: Influenza, A Survey of the Last 50 Years in the Light of Modern Work on the Virus of Epidemic Influenza", by F. M. Burnet and Ellen Clark; 1942. Melbourne: Macmillan and Company Limited. Crown 4to, pp. 126 with 20 illustrations. Price: 17s. 6d. net.

"New Paths in Genetics", by J. B. S. Haldane, F.R.S.; 1941. London: George Allen and Unwin Limited. Demy 8vo, pp. 206. Price: 7s. 6d. net.

"Surgical Instruments and Appliances Used in Operations: An Illustrated and Classified List, with Explanatory Notes", by Harold Burrows, C.B.E., Ph.D., M.B., B.S. (London), F.R.C.S.; Eleventh Edition; 1942. London: Faber and Faber Limited. Crown 8vo, pp. 146, with illustrations. Price: 3s. 6d. net.

Diary for the Month.

AUG. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 5.—Western Australian Branch, B.M.A.: Council.
AUG. 6.—South Australian Branch, B.M.A.: Council.
AUG. 7.—Queensland Branch, B.M.A.: Branch.
AUG. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 11.—Tasmanian Branch, B.M.A.: Branch.
AUG. 14.—Queensland Branch, B.M.A.: Council.
AUG. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 19.—Western Australian Branch, B.M.A.: Branch.
AUG. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 27.—New South Wales Branch, B.M.A.: Branch.
AUG. 27.—South Australian Branch, B.M.A.: Branch.
AUG. 28.—Queensland Branch, B.M.A.: Council.
AUG. 28.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited, all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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